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I

SUPPURATION IN THE PETROUS PYRAMID*

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A routine study of the temporal bones of meningitis cases coming to autopsy has been carried out during the past seven years and forms the basis for the following reports. During this period we have obtained and examined one or both temporal bones from 28 cases of meningitis. This included all of the cases from which this material was available, regardless of the type or the source of the infection.

A considerable number of additional cases were treated during the period, with a fatal outcome, including some cases of petrous pyramid suppuration, but those are not included in this group inasmuch as the pathological material could not be obtained for study.

The origin of the meningeal infection in the 28 cases is indicated in the following table:

T. B. meningitis, with and without middle ear suppuration	5 cases
Osteomyelitis of sphenoid	1 case
Fracture of sphenoid followed by infection	1 case
Frontal sinus infection and bacteremia	1 case
Frontal bone osteomyelitis	1 case
Brain abscess, origin indefinite	3 cases
Torula infection	1 case
Osteomyelitis of cranial vault, otitic	1 case
Meningitis along with acute middle ear suppuration, but the exact relationship not clear	6 cases
Diffuse labyrinthitis and empyema of saccus endolymphaticus	1 case
Suppuration in the petrous pyramid	7 cases

*From the Division of Otolaryngology of the University of Chicago Clinics.

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Out of 28 cases of meningitis were six in which there was an acute middle ear suppuration, but in which the pathway of spread to the meninges could not be determined by the histopathological examination. One of these was a probable petrous apex infection, but the apex was missing from the specimen and a definite diagnosis was not possible. One case, a child of four years, had thrombosis of the right lateral and the superior sagittal sinuses, a bacteremia and suppurating joints, but the right temporal bone was destroyed at the necropsy, and the pathway of extension to the sinus therefore could not be determined.

There were two infants under six months of age, with bilateral middle ear and mastoid antrum suppuration, in whom there was no histopathological evidence of direct extension to the meninges. Although the bone marrow is intimately connected with the antrum mucosa at that age, there was no evidence of an acute osteomyelitic process in the temporal bones, nor was there any absorption of bone in the mastoid.

The remaining two cases out of the six were adults, one of whom had pneumonia as well as the acute middle ear suppuration. In both of these cases, direct extension to the meninges by way of breakdown of pneumatic cells could be ruled out, neither was there any infection of the marrow spaces of the temporal bone.

Of the six cases in which the pathway of extension to the meninges could not be determined from the histopathological examination, two were probably cases of direct extension, while in the remaining four it was possible to rule out both osteomyelitis of the temporal bone, and direct spread by bone resorption. The most likely explanation of the meningitis was a blood stream infection.

One case proved to be an extension from an empyema of the saccus endolymphaticus, while the remaining seven developed meningitis secondary to suppuration in the petrous pyramid. The histopathological findings in these cases will be given in some detail, along with a short clinical history in each case.

The terminology used to describe the different areas of the petrous pyramid still varies with different authors. In the ensuing description the term petrous apex indicates that part of the pyramid medial to, and in front of, the internal auditory meatus. The area posterior to the internal auditory meatus is divided into three posterior cell groups. The posterosuperior area borders on the middle cranial fossa, internal to the arcuate eminence. The posteroinferior area borders on the posterior cranial fossa, posterior to the aqueductus

endolymphaticus. The posterointernal area lies in front of the aqueductus endolymphaticus, and corresponds to the retro-facial group of cells. These posterior cell groups are all perilabyrinthine in location.

CASE 1.—E. B., male, age 22. Onset of earache, left ear, December 9, 1932. Spontaneous discharge began two days later. First vomited December 16, 1932. Retro and periorbital pain were complained of on December 19, 1932. During the next three days, severe headaches, stiffness and pain in back of the neck came on, with temperature ranging to 103°. Admitted December 22, 1932. Slight photophobia, increased knee jerks and plantar extension on the right side. Lumbar puncture; pressure 260. Tobey Ayer test negative. 1600 cells; pneumococcus type III in sediment and on culture. W. B. C. 21,000. X-ray examination showed clouding of mastoid cells on the left, but no changes were noted in the petrous apex, on the occiput-down views. On review of the x-ray plates, subsequent to the histological examination decreased density can be made out on the left. Simple mastoid operation December 22, 1932. Very little cell breakdown except in an area below the antrum which was filled with pus. No fistulæ found. Lateral sinus and dura were both exposed and appeared normal. Meningeal signs increased. Right sixth nerve palsy two days after operation. Died December 27, 1932.

Histopathology—Right temporal bone: Pneumatic mastoid. Posterosuperior and inferior cell areas of the petrous pyramid well pneumatized. Apical portion mostly non-pneumatized. A few large peritubal cells present. Mild inflammatory reaction in the middle ear.

Left temporal bone: Pus and granulation tissue in middle ear and mastoid antrum. The petrous apex is occupied almost completely by a large abscess cavity. Pneumatized spaces extend toward the apex around the superior and posterior canals and through the subarcuate fossa. These spaces mostly contain swollen mucosa and some inflammatory exudate. A fairly well defined tract of pus-containing cells can be seen microscopically extending through the subarcuate fossa to the abscess cavity. Suppuration has eroded into the middle fossa. The greater superficial petrosal nerve and vessels are enclosed by edematous tissue and pus, and lie near the site of erosion of the cortex. A few large pneumatic spaces are recognized in the upper part of the apex, adjacent to the abscess cavity. A few small marrow spaces remain and contain greatly dilated vessels with edematous walls and sparsely distributed young connective tissue. See Figs. 1 and 2.

Summary: Suppuration has reached the apex of the petrosa by way of the pneumatic spaces. The degree of pneumatization of the apex is not clear, but at least there was some pneumatization in the upper part. The abscess represents a coalescence of pneumatic spaces with probably some encroachment on marrow spaces, to form one large cavity, which ruptured through the cortex into the middle fossa. No fistula was found at operation, but a tract of small pus-containing cells is clearly seen in the sections, leading through the subarcuate fossa. Although the organism was type III pneumococcus, there has been extensive bone destruction in the petrous apex, with rupture through the cortex and development of diffuse meningitis within two weeks of the onset of disease.

CASE 2.—E. N., female, age 12. The left ear began to discharge March 28, 1936. Two weeks later, pleurisy and pneumonia were diagnosed and patient

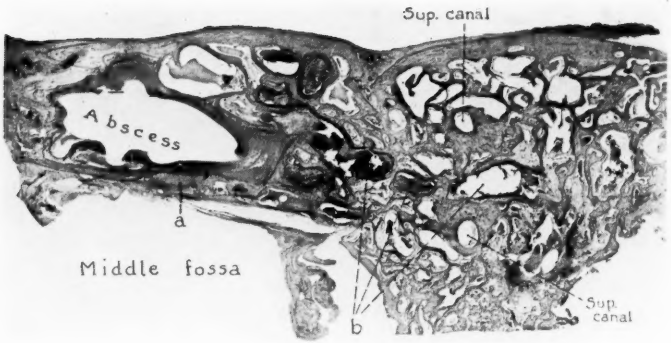

Fig. 1. Photomicrograph showing the abscess cavity in the petrous apex which has ruptured into the middle fossa at *a*. At *b* can be seen a tract of cells extending through the subarcuate region, showing suppuration and necrosis.

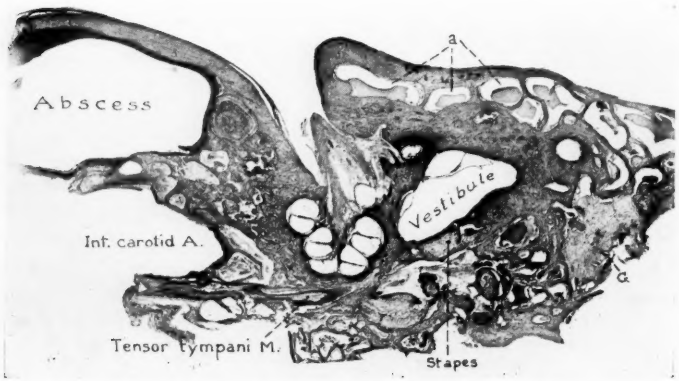

Fig. 2. Photomicrograph at a lower level than Fig. 1, showing the relation of the abscess in the apex to the canal for the internal carotid artery; *a* indicates a tract of cells containing exudate lying posterior to the aqued. endolymphaticus; *g* indicates granulation tissue in the mastoid antrum.

treated at home. Came to hospital May 8, 1936, and was admitted, complaining of pain over the left eye, spreading across the forehead, high temperature and recurrence of aural discharge for three days. W. B. C. 22,000. X-ray showed clouded mastoid cells but no asymmetry of petrous pyramids. Hemolytic streptococcus on culture. Complete mastoidectomy done. No improvement in symptoms. On May 12, 1936, the C. S. F. was cloudy and hemolytic streptococcus present on culture. Tobey Ayer test negative. Died May 16, 1936. Streptococcus meningitis.

Histopathology—Right ear: Pneumatic mastoid. Air cells extend medially above and behind the superior canal and through the subarcuate fossa to the supracochlear region. The apical portion of the bone is missing. No suppuration.

Left ear: Marked swelling and infiltration of the mucosa of the middle ear, with free pus. There are several large pneumatic spaces in the posterosuperior cell area, which have developed from the mastoid and from the attic. These cells contain greatly thickened and infiltrated mucosa and some exudate. New bone is being deposited on the walls of several of these spaces. The apical portion of the petrosa is not all present. The inferior part, extending from beneath the cochlea anteriorly, is occupied by a mass of granulation tissue, in the center of which is an abscess cavity. The swollen epithelium lining the cavity is still evident. In the deeper layers of the granulation tissue are many trabeculae of new bone. The marrow spaces in the upper part of the apex show marked reaction. This consists mainly of newly formed fibrous connective tissue arranged along the bony walls and trabeculae, and around blood vessels, as well as vascular dilatation and infiltration of plasma cells, macrophages and small lymphocytes. The bony cortex over the jugular bulb has been eroded away. The vessel itself is not present in the preparation. A tract of diseased pneumatic cells extends from the hypotympanum forward beneath the cochlea to the site of the abscess in the apex. (See Figs. 3 and 4.)

Summary: The apical portion of the petrous pyramid has been penumatized in the inferior part. The pneumatic cells in the posterosuperior part of the pyramid show some new bone formation but do not connect with the abscessed area in the apex. Suppuration has extended by way of pneumatic spaces forward from the hypotympanum into the apex, producing a coalescent osteitis with abscess formation. The bone marrow in the superior half of the apex shows a marked degree of reactive fibrosis. The sections show that the abscess in this case could not possibly have been reached from the simple mastoid operation. The intrapetrosal approach to the apex from the mouth of the eustachian tube, through the carotid canal, would have led into the suppurating area; also, since the abscess was in the inferior part of the apex, the extra petrosal approach to the inferior surface might have been successful.

CASE 3.—E. A., female, age 42. Specimen sent for examination from another hospital with the following history: Right earache began March 12, 1935. Paracentesis four days later. Mastoid operation March 17, 1935. Discharge from hospital March 24, 1935, on the second day without fever. Complained of headache March 23, 1935, which continued, localizing about her right eye and cheek and extending to the vertex. April 7, 1935, paralysis of right sixth and seventh nerves. Mastoid wound reopened and explored. Died April 13, 1935. Meningits. Culture not reported.

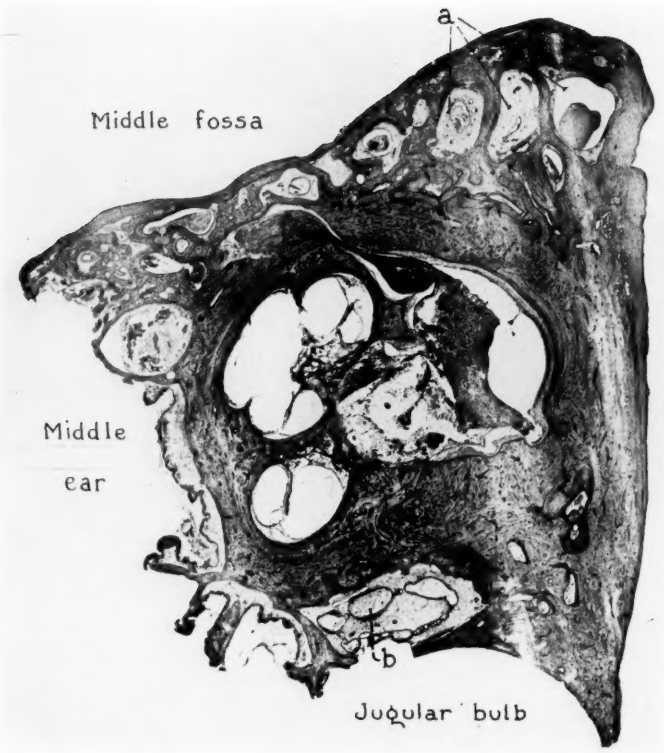

Fig. 3. Photomicrograph of vertical section through the region of the cochlea (case 2). At *a* can be seen inflammatory reaction with some osteogenesis in pneumatic cells in the posterosuperior angle. At *b* can be seen the beginning of a cell tract leading to the petrous apex.

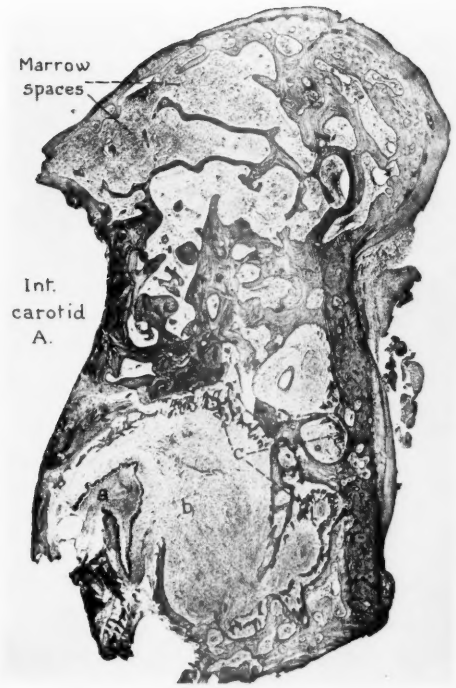

Fig. 4. Photomicrograph of a section anterior to the cochlea (case 2); *a*, epithelium lined cavity filled with pus; *b*, granulation tissue; *c*, new bone which has been laid down along the margins of the suppurating focus. The marrow spaces in the upper part of the section show a moderate degree of fibrosis.

Histopathology: Only the right temporal bone was obtained. Middle ear mucosa greatly swollen and infiltrated. Mastoid cells contain exudate and have greatly thickened mucosa. Pneumatization of the petrosa is extensive, with tracts of cells extending forward above and behind the labyrinth and through the subarcuate fossa. Some pneumatic spaces are recognized in the apical portion, as well as some marrow spaces, but the apex is mainly taken up by a large abscess cavity surrounded by granulation tissue. The bone in the apex has been extensively destroyed. The remaining marrow spaces show a marked irritative reaction with proliferation of young fibrous tissue and new bone being laid down. The pneumatic spaces extending through the subarcuate fossa show more intense inflammatory reaction than the other mastoid cells, suggesting that this has been the pathway by which the infection reached the apex. There is no fistula, however. (See Figs. 5 and 6.)

Summary: Suppuration extended to the petrous apex, probably by way of pneumatized tracts, from the mastoid. An abscess formed in the partly pneumatized apex. The clinical course of the case, as has been outlined above, was typical of suppuration in the petrous apex. On the basis of the postmortem findings, it is evident that drainage of the empyema via the mastoid would have been at least very difficult. The intrapetrosal approach from the mouth of the eustachian tube would seem to have been the most logical method to obtain drainage.

CASE 4.—K. A., female, 58. Diabetes of 14 years duration. Bilateral otitis media, suppurative, came on 11 weeks before admission. The patient was hospitalized elsewhere and the diabetes treated by means of insulin. The ears improved and the right stopped discharging. She went home from the hospital apparently much improved. Three days before admission to this hospital the left ear stopped discharging and she became acutely ill. Two days later she was again able to be up and about in the morning, but in the afternoon became rapidly worse, complaining of headache and vomiting several times. That evening she was admitted in a moribund state with signs of meningitis. Spinal fluid contained 8,300 cells and type III pneumococcus. Died within 24 hours. An accurate history of the illness previous to her admission here could not be obtained.

Histopathology—Right ear: The middle ear spaces and mastoid cells show a subsiding inflammatory reaction. The middle ear contains exudate, which is almost free from cells, and a moderately thickened mucosa. The mastoid antrum and adjacent air cells are filled with granulation tissue, and more outlying cells show only a mild reaction with some exudate and thickened mucosa. There has been some bone absorption in a few areas. The posterior cell group in the petrosa are well pneumatized. No evidence of osteogenesis. (See Fig. 7.)

Left ear: Much of the temporal bone is missing from the specimen. The remaining portions show the mastoid antrum filled with granulation tissue and some exudate. There is extensive pneumatization and most of the cells have a greatly swollen mucosa with irregular absorption of bony partitions. In the posterior part of the mastoid there has been breaking down of cell walls to form a tract leading to an abscess cavity lying in the petrosa, external to the aqueductus endolymphaticus. The abscess has formed in pneumatic spaces, the result of absorption and breakdown of cell partitions. The walls of the abscess are made up of thick granulation tissue containing small sequestra. In the deeper layers there has been extensive new bone formation. (See Fig. 8.)

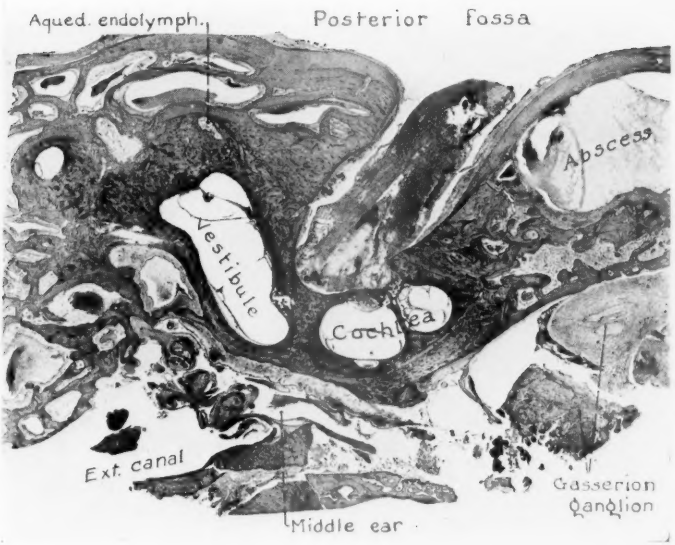

Fig. 5. Photomicrograph of a horizontal section (case 3). A large abscess occupies the posterior part of the petrous apex. A tract of pneumatic cells filled with exudate extends medially behind the aqued. endolymphaticus.

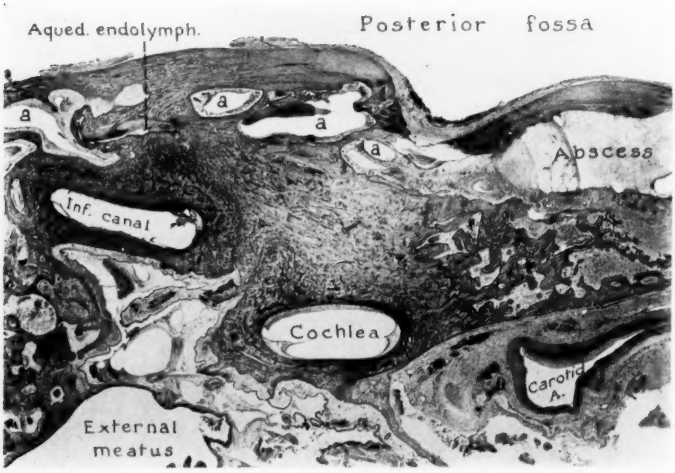

Fig. 6. Photomicrograph of a horizontal section (case 2) to show the relation of the abscess in the apex to the canal for the carotid artery, the cochlea and the tympanic orifice of the eustachian tube.

Summary: The specimen shows an abscess in the posterior inferior cell group of the petrosa, which had eroded into the posterior fossa. An accurate history of the symptoms could not be obtained, but the otitis had been present for 11 weeks. The patient was moribund when admitted, but it is evident that the suppurative focus had been localized for some time, and that if a careful mastoid exenteration had been done a tract could have been located leading to the abscess.

CASE 5.—P. G., female, age 6. History of left otitis media suppurative for five weeks. Vague headaches began in the fourth week and two days later became severe, located in left eye and down the back of the neck, intermittent in character. Irregular fever was present since the onset of the otitis. Patient was hospitalized, two spinal punctures made, and hemolytic streptococci were said to have been cultured from the fluid. Admitted to this hospital March 28, 1936. Definite signs of meningitis present. Stiff neck, positive Kernig's sign, severe headache and irritability, Babinsky sign negative bilaterally. Spinal fluid contained 4,000 cells per ccm., 86 per cent polymorphonuclears, normal pressure. Tobey Ayer test negative. W. B. C. 38,000. Simple mastoid operation was done March 28, 1936. Pus and cell breakdown in the antrum region, extending down toward the tip. No sign of fistula leading to deeper parts of the bone. The course of the illness was only slightly improved following the operation. Hemolytic streptococcus cultured from C. S. F. April 2, 1936. Death April 8, 1936.

Histopathology—Right ear: The middle ear spaces are free from suppuration. The apex of the petrous pyramid is not pneumatized. It is made up of a hematopoietic bone marrow. There are a number of large pneumatic spaces in the posterior-superior and inferior cell areas of the pyramid, which are a direct extension both from the epitympanic space and from the mastoid, above the superior canal.

Left ear: The middle ear and mastoid cells show a moderate inflammatory reaction. The pneumatic spaces in the posterosuperior cell group have greatly thickened, infiltrated mucosa, with much absorption of bone from the walls. In several areas the inflammatory reaction and bone absorption has extended through the cortex to the middle fossa. This extension has apparently followed already existing vascular channels which are irregularly broadened, by absorption of the bony walls, and contain some loose connective tissue and mononuclear cells. There has been considerable osteogenesis and fibrosis in the deeper layers of the mucosa and there is some erosion as well as an irrgular layer of new bone, laid down on the superior surface of the pyramid, outside the dura. A tract of pneumatic cells, which contain some exudate, extends from the medial wall of the attic in the cell area in front of the arcuate eminence. There is no connection to be seen with the mastoid cavity. No pneumatization is seen in the apex, but both fatty and hematopoietic marrow is seen. These marrow spaces show a moderate inflammatory reaction. This is most recognizable in the fat marrow where there is hyperplasia of fibrous connective tissue, dilatation of vessels and infiltration by plasma cells and macrophages. (See Figs. 9, 10 and 11.)

Summary: Suppuration has extended inward from the attic to the air cells, medial to the arcuate eminence. Erosion of the cortex has taken place, with extension to the middle fossa. The process has apparently been present beneath the dura for some time, as considerable osteogenesis has occurred. There is no cellular connection with the mastoid in this case and no fistula from the middle ear, but it would have been possible to have reached the diseased area by elevating the dura medial to arcuate eminence.

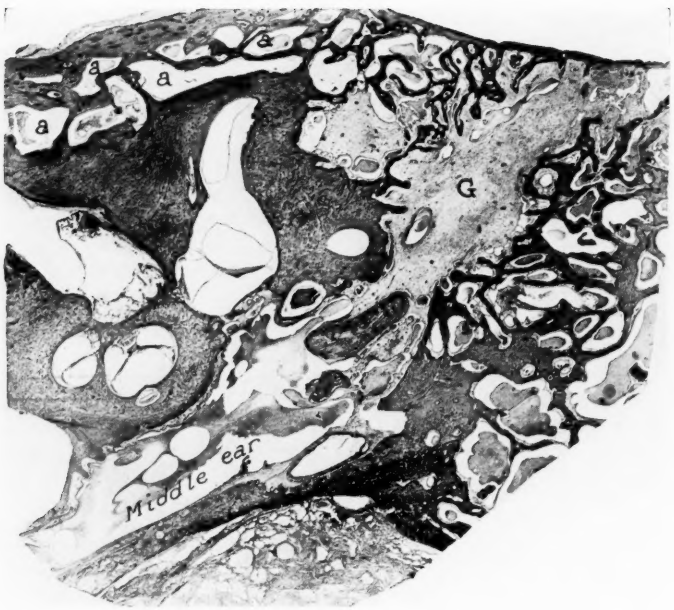

Fig. 7. Photomicrograph of a horizontal section through the right ear, showing a healing mastoiditis.

a. Pneumatic cells extending posterior to the labyrinth.

G. Granulation tissue in the mastoid antrum.

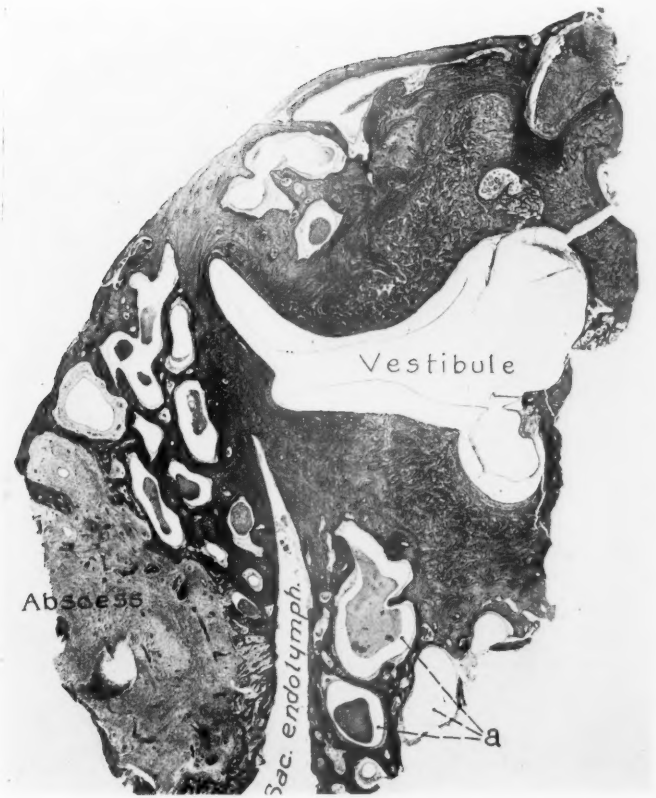

Fig. 8. Photomicrograph of a vertical section through the left ear showing the abscess cavity external to the aqued. endolymphaticus which has ruptured into the posterior fossa. *a*. Pneumatic cells of the postero-internal cell group, containing exudate.

CASE 6.—J. P., male, age 6. Admitted to hospital April 29, 1933, with the following history: Two months before admission child had scarlet fever from which he recovered in about one month. Tonsils and adenoids were then removed. He recovered from the operation and felt fairly well except that he had lost weight consistently since the onset of the scarlet fever. On April 29, 1933, he began to vomit and couldn't retain food. On April 29, 1933, a lumbar puncture was done and 800 cells per cc. found in the fluid, along with many organisms which were said to be Gram negative diplococci. Antimeningococcus serum was given and patient was then transferred to this hospital. On following day the C. S. F. was found to contain hemolytic streptococci in large numbers. Subsequent questioning elicited the information that the boy had complained of earache following the scarlet fever but the otitic infection had been overlooked. Died of streptococcus meningitis May 3, 1933.

Histopathology—Left ear: Low grade inflammatory reaction in mucosa of middle ear and mastoid cells. The posterior-superior cell area of the petrous pyramid is pneumatized.

Right ear: Moderate thickening and infiltration of mucosa in the middle ear and mastoid cells. Medial to the arcuate eminence is a group of cells which show a marked inflammatory reaction, with extensive absorption and regeneration of bone. Erosion of the cortex has occurred, and new bone has been laid down on the floor of the middle fossa and on the walls of the internal meatus. A well defined microscopic tract extends from the attic to this area of coalescence, with active absorption and regeneration of bone. Within this area there is a cyst-like structure occupying most of the space, consisting of a central area or lumen filled with homogeneous fluid and surrounded by a wall composed of fibrin, outside of which are layers of young fibrous tissue, continuous with the fibrous tissue which infiltrates the whole diseased area. From the lower part of this area inflammatory reaction can be traced both to the internal meatus and to the posterior fossa, along the vascular channels in the bone. The bony walls of these channels (Haversian canals) have been eroded and young connective tissue fills the spaces. The bone marrow of the apex shows a marked reaction. There are dilated vessels, proliferation of fibrous tissue and infiltration by macrophages, plasma cells and lymphocytes. New bone has been laid down in marrow spaces adjacent to the focus of bone absorption. (See Figs. 12, 13 and 14.)

Summary: An area of suppuration and cell breakdown (coalescent osteitis) was found medial to the arcuate eminence. This had extended by way of pneumatic spaces from the attic and had eroded to the middle fossa. A reactive fibrosis was found in the marrow spaces of the apex. A fistulous tract was found connecting the epitympanic space. The presence of an otitis media had been overlooked, but it is evident that had a mastoid operation been done, it would have been necessary to expose and elevate dura beyond the arcuate eminence, or approach the focus from the attic region, in order to effect drainage.

CASE 7.—J. M., male, age 5½. The child was brought to the clinic December 9, 1930, with complaint of total loss of hearing and severe headache. The history was as follows: About November 1, 1930, onset of hearing impairment, earache and discharge right ear with vomiting. One week later tonsils and adenoids were removed by his physician. No improvement in his condition for the next two weeks. November 22, 1930, sudden onset of violent dizziness and vomiting, lasting five days. Some improvement until December 5, 1930, and then began

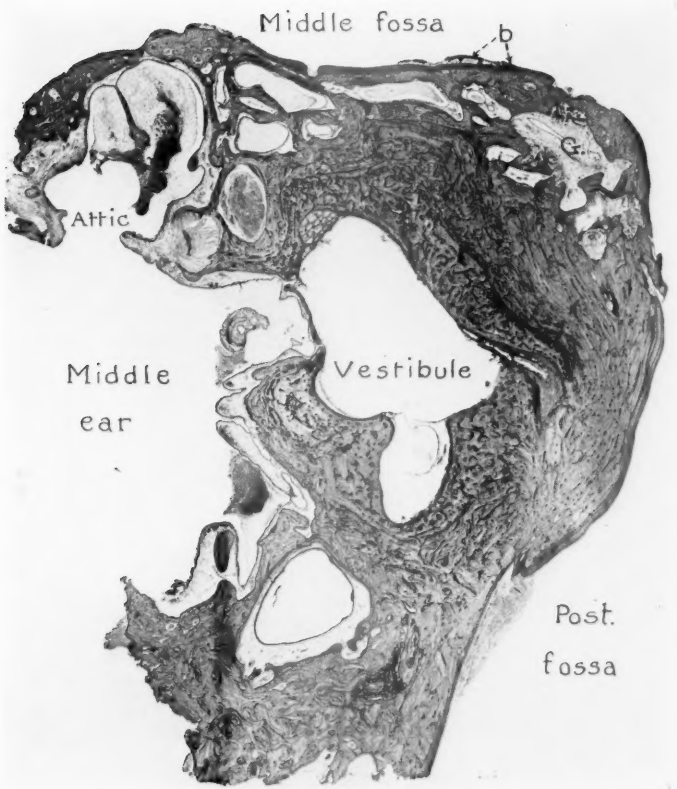

Fig. 9. Photomicrograph of a vertical section through the vestibule of the left ear (case 5). *G.* Granulation tissue, bone absorption and osteogenesis in pneumatic cells of the posterosuperior angle, medial to the arcuate eminence. *b.* New bone which has been laid down on the floor of the middle fossa.

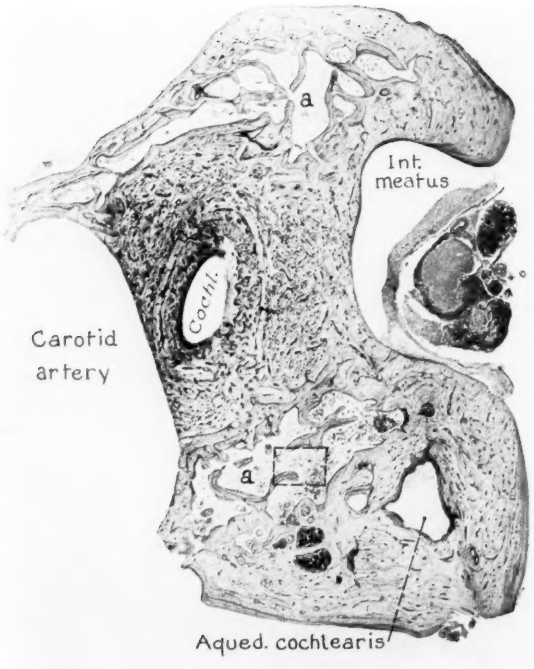

Fig. 10. Photomicrograph of vertical section through the anterior edge of the cochlea (case 5) to show the early stages of a reactive fibrosis in the bone marrow. *a.* A higher magnification of the "inset" area is shown in Fig. 11.

to vomit again. On December 7, 1930, the child became completely deaf. Vomiting continued. Some fever noted. Admitted December 9, 1930. Definite signs of meningitis. Mastoid x-rays showed clouding of both mastoids, more so on the right. Temperature around 39°C. W. B. C. 22,000, with 80 per cent polys. No hearing demonstrable in either ear. Right facial paralysis. Both ear drums intact, the right dull and full. Right mastoid exenteration December 10, 1930. Pus in the antrum and some cell breakdown. Left mastoid operation December 11, 1930. Cells contained edematous mucosa throughout but no pus. The dura of middle and posterior fossa was widely exposed on both sides by operation. Type I Pneumococcus was isolated from the C. S. F. December 17, 1930. Death from meningitis. Autopsy five hours later. On removal of the temporal bones from the skull, an abscess cavity was found beneath the right pyramid, extending down along the posterior belly of the digastric and stylohyoid muscles.

Histopathology: Both temporal bones were accidentally sectioned in the longitudinal vertical plane.

Right ear: Middle ear mucosa thickened and infiltrated and the cavity filled with pus. Both the round window membrane and the annular ligament are infiltrated with leucocytes. The greater part of the apex of the petrous pyramid is missing, but some peritubal cellular spaces are seen which have a greatly thickened lining mucosa and contain purulent exudate. The marrow spaces of the apex show marked edema and dilatation of blood vessels with a loss of the normal structure and replacement by an irregularly distributed exudate which in some areas is infiltrated by round cells and in some areas is purulent. In the superior part of the apex, close to the internal meatus, there is a small abscess cavity of which only one border is preserved. This cavity lies posterior and medial to the cochlea, bordering on the middle fossa and anterior border of the internal meatus. Between the marrow spaces and the abscess cavity irregular deposits of new bone have been laid down, also in the internal meatus. The abscess opens freely into the middle fossa. Posterior to the internal meatus there are several pneumatic spaces, which have extended medially from the mastoid, filled with exudate. The walls of these spaces are greatly thickened and some osteogenesis is present in the deeper layers. (See Figs. 15 and 16.)

Left ear: The middle ear and mastoid spaces are similar to the right. There is pus in the middle ear and in some of the mastoid cells. A few pneumatic spaces, containing purulent exudate, are seen in the peritubal region. The petrous apex is not pneumatized, but a few pneumatic cells are seen medial to the arcuate eminence. The labyrinth spaces are filled with pus. In several areas one sees a localized area of erosion of the endosteal and enchondral layers of the labyrinth wall, the eroded space filled with pus, dilated vessels, and young fibrous tissue. Irregularities in the walls are seen resembling lacunae, but seldom containing osteoclasts. The bone of the capsule is riddled in many places by areas of absorption, corresponding to vascular spaces in the bone, which have been enlarged and contain young fibrous tissue. No evidence of osteogenesis. The erosion along vascular spaces is seen in all layers of the bony capsule. The marrow of the apex has greatly dilated and edematous vessels and venous spaces. The normal elements of bone marrow have been replaced by an inflammatory exudate, which in some areas is frankly purulent. No evidence of any regeneration of bone. (See Figs. 17 and 18.)

Summary: The correlation of the histopathological findings with the clinical history in this case indicates that the sequence of events has been as follows:

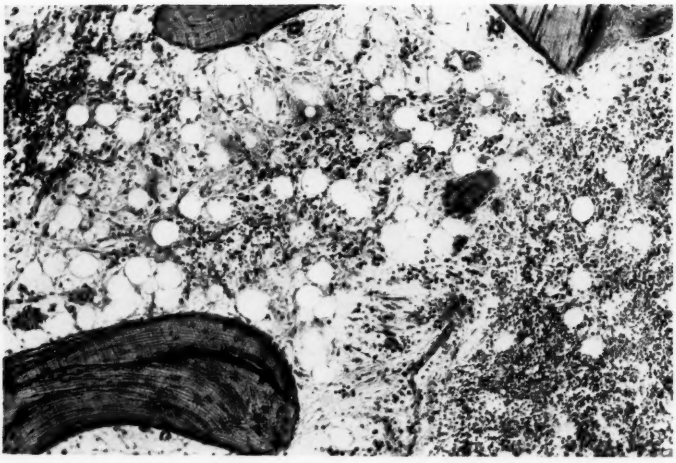

Fig. 11. Photomicrograph of the area of bone marrow indicated in Fig 10, showing dilated vessels, young fibrous connective tissue and cellular infiltration (macrophages, plasma cells and lymphocytes) involving fatty marrow and red marrow alike.

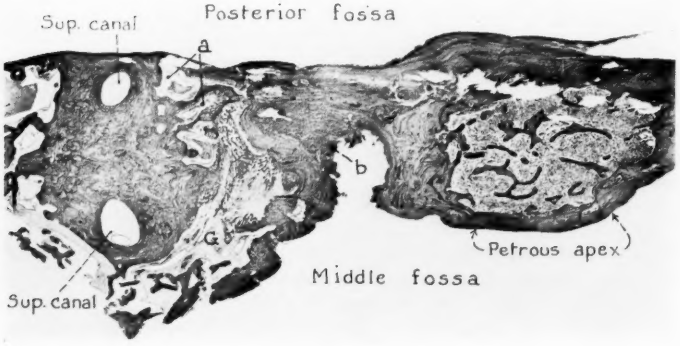

Fig. 12. Photomicrograph of a horizontal section of the right ear (case 6); *G* indicates a tract of suppuration extending from the attic to the pneumatized area (*a*), medial to the arcuate eminence. New bone has been laid down in this tract as well as in the floor of the middle fossa (*b*).

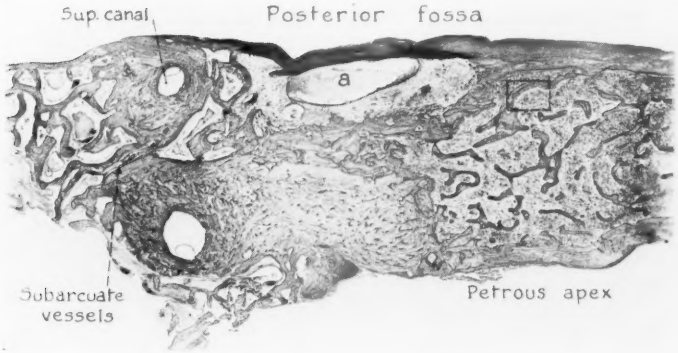

Fig. 13. Photomicrograph of a section at a lower level (case 6). *a*. Cyst-like cavity within the area of coalescent osteitis. A marked fibrosis is seen throughout the bone marrow of the petrous apex. A higher magnification of the "inset" area is shown in Fig. 14.

Fig. 14. Photomicrograph of the area shown in Fig. 13. An advanced degree of fibrosis (in comparison with that shown in Fig. 11) is seen and a thick layer of new bone has been laid down in apposition to one of the bony trabeculae of the marrow spaces.

A right otitis media and mastoiditis occurred, followed by infection in retrolabyrinthine cells, and extension to the apex. A localized abscess formed in the apex, either as a localized osteomyelitis or an extension into pneumatic cells which were later obliterated, and ruptured into the middle fossa. The right sided labyrinthitis may have been a spread from the disease in the bone or may have been secondary to meningitis. The left sided labyrinthitis occurred late in the disease as a spread from the meninges. The diffuse bone destruction and middle ear changes on the left are apparently secondary to the labyrinth suppuration. In addition to the localized abscess in the right apex which had been present long enough for osteogenesis to occur, there is a bilateral acute osteomyelitis of both apices, more marked on the left side, which presents the appearance of an acute fulminating process and has probably developed only in the late stages of the illness.

Case 8.—M. D., male, age 56. On January 24, 1931, during a severe head cold, he began to have roaring and pain in the right ear. A myringotomy was done. Two days later he developed severe vertigo, nausea and vomiting, and a tendency to fall backwards. The discharge was scanty. During the next week the symptoms decreased steadily. No fever. Discharge decreasing. On February 11, 1931, he came into the hospital with severe headache over the vertex, of 12 hours duration. Definite signs of meningitis were present. Type III pneumococci were present in the spinal fluid, with increased pressure and high cell count, mostly polymorphonuclears. Patient was not known to be a diabetic, but had sugar in the urine. Died February 12, 1931. Pneumococcus type III meningitis.

Histopathology—Left ear: No suppuration in middle ear spaces. The mastoid is pneumatic but no air cells are present in the pars petrosa. There is an area of otosclerotic bone at the oval window.

Right ear: Marked inflammatory changes in the middle ear with extension through the oval window. The vestibule is filled with pus. Granulation tissue fills the antrum and the surrounding mastoid cells show thickened mucosa and exudate. No pneumatization of the apex or of the posterior cell areas in the pyramid. The aqueductus endolymphaticus and the saccus are filled with pus which has invaded the walls and extended directly through the dura covering the saccus. The dura is edematous. The internal auditory meatus contains considerable purulent exudate. The labyrinth capsule, particularly the enchondral layer, is riddled by bone absorption and proliferation of young connective tissue which extends along vascular channels. This is most evident in the walls of the saccus and aqueductus endolymphaticus.

Summary: Acute otitis media in a diabetic. Pneumococcus type III. Acute diffuse labyrinthitis developed within two days of the onset. An abscess developed in the endolymphatic sac and the suppuration extended directly through the dura with a fatal termination in 19 days. The sections demonstrate how suppuration may pass from a localized focus directly through the swollen dura. The clinical history and the pathological findings indicate that the infection had remained localized during the early stages of the disease, and that had the proper time been selected, drainage of the labyrinth and saccus endolymphaticus probably might have been successful. Without entering further on a discussion of the treatment of labyrinthitis, it may be said that the case illustrates the necessity of maintaining close observation during the disease so that surgical intervention may be carried out promptly on the appearance of any meningeal signs. (See Figs. 19 and 20.)

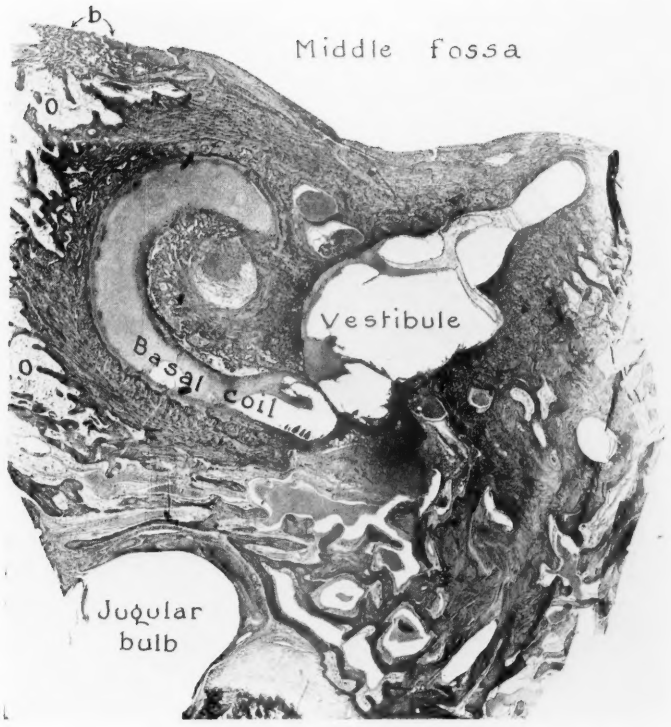

Fig. 15. Photomicrograph of a vertical longitudinal section of the right temporal bone (case 7). The labyrinth spaces contain pus throughout. The middle ear spaces are filled with inflammatory exudate. An acute inflammatory reaction is present in the marrow of the apex (*o*). Above the cochlea can be seen a mass of new bone (*b*) which borders on an abscess cavity. This area is better shown in Fig. 16.

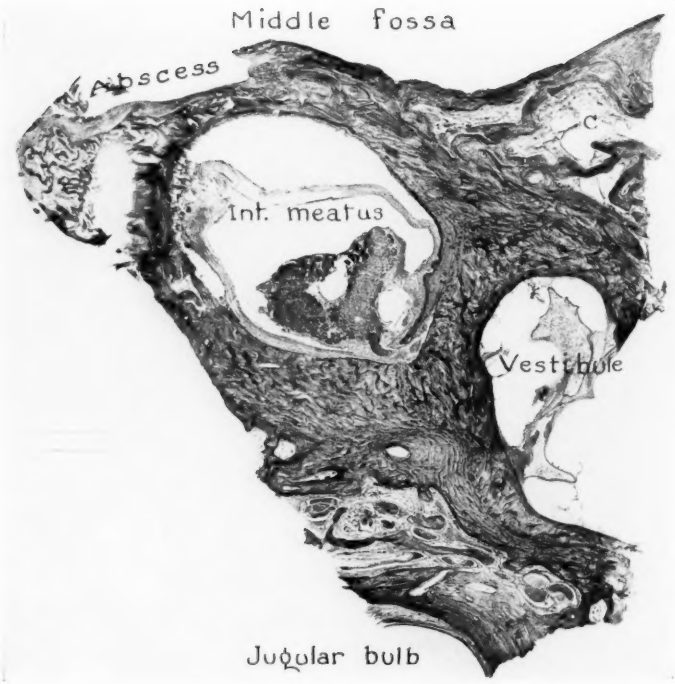

Fig. 16. Photomicrograph of a section posterior to Fig. 15. An abscess cavity is shown above and medial to the internal auditory meatus, opening into the middle fossa. Considerable osteogenesis is seen in the abscessed walls and outside the dura of the internal meatus. A group of pneumatic cells (*c*) is present above the vestibule. The lining of these cells is greatly thickened, with some osteogenesis in the deeper layers. These pneumatic cells were an extension from the mastoid.

Fig. 17. Photomicrograph of a vertical longitudinal section through the left ear (case 7). A diffuse purulent labyrinthitis is shown, and at (*a*) extensive absorption of the bony capsule has taken place, extending outward through the dense enchondral layer. (*c*) Some pneumatic cells which have extended medial to the arcuate eminence. The bone marrow of the apex (*o*) shows an acute diffuse osteomyelitis. A higher magnification of the inset area is shown in Fig. 18.

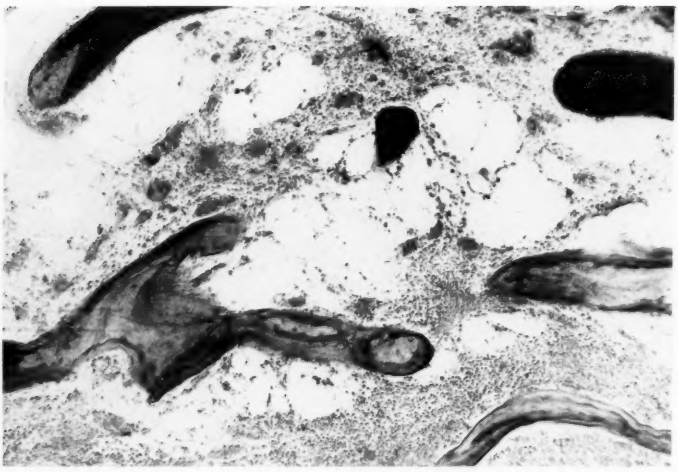

Fig. 18. Photomicrograph of the bone marrow of the apex (case 7) showing early acute osteomyelitis. There is marked dilatation of the vascular space with edema. A diffuse cellular infiltration has taken place which in some areas consists almost entirely of polymorphonuclear leucocytes. There is little evidence of bone absorption and no sign of new bone formation.

CASE 9.—D. W., male, age 10. About November 26, 1936, the left ear began to discharge following a severe cold. He was treated at home. January 11, 1937, he began to have severe pain in the left temporal region and above the left eye and vomited several times during the following week. His temperature was said to be normal. Admitted to hospital January 18, 1937. His chief complaints were of pain over the outer half of the left supraorbital ridge and profuse discharge from the left ear. He had normal temperature. W. B. C. 11,700. Culture from the ear, hemolytic streptococcus. Neurological examination negative except for positive Babinski's reflex on right side.

Mastoid x-rays showed diffuse clouding of the mastoid cells on left. Petrous pyramid views are indefinite. The simple mastoid operation was done on January 18, 1937. He had a pneumatic type of mastoid, with considerable sclerosis. Some pus in cells in the tip and in the antrum region. No evidence of tract leading towards the petrosa. After operation the pain persisted above the left eye, and on January 20, 1937, lext sixth nerve palsy appeared. X-rays of petrous pyramids were repeated. The right apex appeared pneumatized, while on the left no cell partitions could be seen and the density was decreased. The C. S. F. cell count was normal and the Tobey Ayer test negative. January 21, 1937, revision of mastoid wound. The radical mastoid operation was done. No fistula seen. The petrous apex was explored by means of small curette between the cochlea and the internal carotid. Several cc. of thick pus were evacuated from the apex. Profuse discharge from the middle ear with occasional remissions of pain over the left eye persisted during the next six weeks. March 10, 1937, removal of granulations from the middle ear and completion of the radical mastoid operation. There was an uneventful recovery, with gradual disappearance of the abducens palsy. The ear is dry at the present time and the whispered voice can be heard at one to two feet.

Summary: The diagnosis in this case presented no difficulty, inasmuch as the history, the location of the pain, the x-ray findings and the appearance of the abducens palsy all indicated infection within the petrous apex. The positive Babinski reflex on the right side, with normal cerebrospinal fluid, was a significant finding indicating that an inflammatory reaction was present in the meninges over the apex. The temperature was never elevated more than a degree during his stay in the hospital. The x-ray plates showed a trabecular arrangement of the bone in the healthy apex, indicating pneumatization, and it is probable that the abscess in the diseased apex resulted from suppuration in pneumatized spaces.

CASE 10.—J. L., female, age 8. About March 1, 1937, left ear began to discharge following a severe cold of two weeks duration. In a few days she began to have pain over the left eye. The temperature reached about 101° F daily and there was slight vertigo at times. She lost 15 pounds in four weeks. Admitted March 30, 1937. Profuse purulent discharge from the left ear. Severe intermittent pain over the outer border of left supraorbital ridge. Positive Babinski's reflex on right side. Other neurological examinations negative. The x-ray showed clouded mastoid cells on the left side and an asymmetry of the petrous apices. The left was more dense than the right. March 30, 1937, simple mastoid operation. The cells were not broken down but were filled with thickened mucosa and granulation tissue. No fistulæ found. April 1, 1937, pain over the left eye had persisted. Paresis of left external rectus muscle appeared. Temperature about 102° F. daily. No chills. The radical mastoid operation was done, with removal of the anterior buttress and the anterior canal wall. Exploration

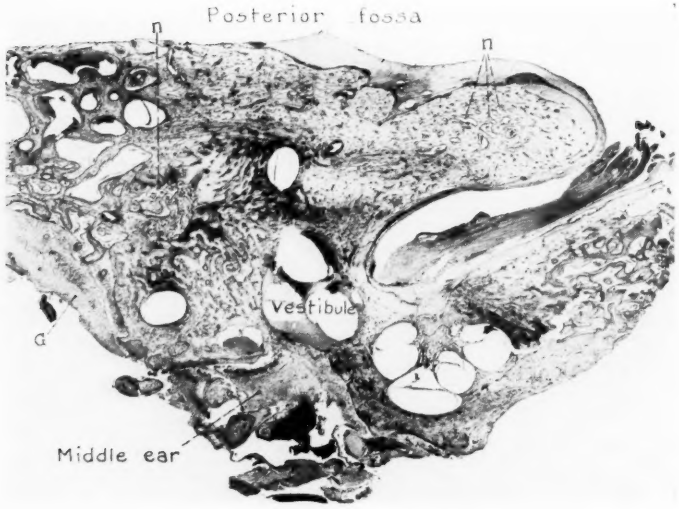

Fig. 19. Photomicrograph of a horizontal section through the right ear (case 8). A diffuse labyrinthitis is shown. *G*. Granulation in the mastoid antrum. At *n* can be seen small areas of bone absorption, extending mainly along vascular channels.

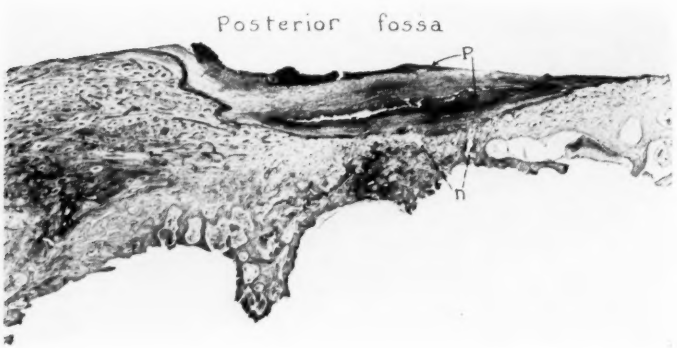

Fig. 20. Photomicrograph through the saccus endolymphaticus (right ear, case 8). Pus can be seen filling the saccus and extending through the edematous layers of its walls to invade the posterior fossa; *n* indicates extensive absorption of bone of the capsule around the saccus.

of the petrous apex was done after exposing the carotid artery and about 5 cc. of pus liberated from an abscess cavity in the apex. The sigmoid sinus appeared normal at operation. Spinal tap showed clear fluid. Normal cell count. Tobey Ayer test showed a block on the left side. April 3, 1937, temperature rose to 39.3°C. The left jugular was ligated. Needle puncture of the sigmoid sinus revealed free bleeding and the sinus was not opened. Blood transfusion of 200 cc. given. Pain recurred at intervals for several days. Paralysis of sixth nerve slowly disappeared. Uneventful recovery.

Summary: The case presented a clear-cut syndrome of suppuration in the apex of the pyramid, namely, pain over the eye, sixth nerve palsy, and x-ray evidence of disease in the apex. The spinal fluid was clear, but plantar extension was present on the right side. The high fever and positive Tobey Ayer test were probably due to thrombosis of the jugular bulb as a result of the suppuration in the apex. Drainage of the empyema of the apex was probably of more importance in the treatment of the thrombosis than was the ligation of the jugular, inasmuch as the recovery followed without opening the sinus. The x-ray plates showed some evidence of pneumatization in the right petrous apex, indicating that the process on the left side was probably an extension into a pneumatized apex.

CASE 11.—K. McM., male, age 6. The right ear began to discharge on June 15, 1937, following a severe cold. Admitted June 25, 1937. There was profuse discharge from the ear and temperature 106.2° F. (R). The x-ray showed the right mastoid cells clouded and right petrous apex more dense than the left. W. B. C. 17,600. C. S. F. clear. Tobey Ayer test normal. A simple mastoidectomy was done on June 25, 1937. The mastoid cells were not broken down but were filled with purulent exudate and granulations. The dura over the lateral sinus and middle fossa appeared normal. Streptococcus hemolyticus on culture. A septic temperature continued during the next ten days. There was some headache, right side, not localized. W. B. C. increased to 25,000. July 3, 1937, the blood culture was positive for hemolyticus streptococcus. July 4, 1937, spinal tap, clear fluid. Negative Tobey Ayer test bilaterally. Revision of mastoid wound. Lateral sinus exposed widely and appeared blue and smooth on the surface. The radical mastoid operation was done and the petrous apex explored. A very small amount of pus was found. Septic temperature continued and signs of jugular thrombosis became definite. July 6, 1937, ligation of thrombosed jugular and removal of clot from sigmoid sinus were carried out. Blood transfusions were given July 2, and July 9, 1937. Prontylin was started by mouth on July 7, 1937, and continued until July 22, 1937. An uninterrupted recovery followed.

Summary: The presence of infection in the petrous apex was indicated by the location of the pain and the x-ray appearance. Insufficient time had elapsed for breakdown of bony trabeculæ, and the x-ray therefore showed the diseased apex to be more dense than the normal (in contrast to case 9 where there was decreased density and loss of bony cell partitions). The thrombosis of the jugular bulb can best be interpreted as a result of the apex infection, but in view of the fact that the bacteremia was present several days before the interruption of circulation in the jugular, it seems likely that the thrombosis started in the inferior petrosal vein. Considering the case in retrospect it must be admitted that recovery might have occurred after jugular ligation and removal of the clot from the sinus. Against that must be weighed the definite knowledge that jugular

thrombosis may extend from an apex infection, and in such a case ligation and removal of the clot may not prevent a meningitis if the focus in the petrous bone is not drained.

DISCUSSION

PATHOLOGY

Meningitis of otitic origin is frequent in relation to meningeal infections of other origin, as is illustrated in the table given above. Out of 28 cases of meningitis of all types from which the necessary pathological material could be obtained 15 occurred in the course of acute otitis media. The next most frequent type was tuberculous meningitis of which there were five cases in the groups, while frontal and sphenoid sinus infection each accounted for two additional cases.

The route by which the infection traversed the temporal bone and invaded the meninges could be traced definitely in nine of the fifteen cases, by the histopathological examination. Two of the cases could not be considered because the parts of the temporal bones removed at autopsy were inadequate for study. In the remaining four cases it was possible to rule out any direct spread of suppuration through the petrous pyramid, either by pneumatic spaces or through bone marrow.

It seems probable in these four cases that the meningeal infection may have been hematogenous in origin, and in two of them a concurrent pulmonary infection was an equally probable source for a blood stream invasion. Of the remaining nine cases of otitic meningitis, one case, that of a diabetic with osteomyelitis of the squamous part of the temporal bone, is not described in this communication since the petrous was not involved. Case No. 8, with acute labyrinthitis and empyema of the saccus endolymphaticus is included with the group to illustrate one of the relatively less common routes by which infection reaches the meninges, and to demonstrate how suppuration outside the dura may extend to the subarachnoid space, a link in the chain which is frequently not seen in the histopathological examination. In this case suppuration is seen extending directly through the swollen edematous wall of the dura. The remaining seven cases illustrate a number of pathological features which may be briefly discussed.

In this series of cases it was found that the predominating pathological lesion in every instance, with possibly one exception, was a suppurative process within pneumatic spaces (coalescent osteitis), which had produced destruction of cell partitions and had eroded

through the cortex either directly or along preformed vascular channels in the bone, to the middle or posterior cranial fossa or both. In nearly every instance considerable osteogenesis had occurred in granulation tissue, both in the lining of infected pneumatic spaces, and in adjacent marrow spaces, also in the granulating walls of the abscess cavity, and on the inner surface of the bony cortex beneath the dura. The one possible exception is seen in case 7 where a localized abscess was found, apparently in bone marrow. However, in close proximity to the abscess are a group of pneumatized spaces with a marked inflammatory reaction and osteogenesis within their walls, and it is not possible to determine from the available material whether the abscess represents a localized osteomyelitis or whether it had occurred in pneumatic spaces which were later obliterated. The most significant feature was the evidence that the suppuration had remained localized for a considerable period before the development of the diffuse meningitis or the panotitis, and might during that time have been amenable to surgery.

The effect of a closed or confined focus of suppuration upon the bone marrow is illustrated in several of the above cases. It is well known that there is free communication between marrow spaces and the mucosa of pneumatic cells and the mastoid antrum in the young child, but in the adult the intervening wall of the bone is more complete and probably provides a more effective barrier to infection. In the presence of a confined focus of suppuration in either the retrolabyrinthine or apex cells the bone marrow of the apex usually showed a definite reaction. This reaction may be described as a fibrosis, which starts along the periphery of the marrow spaces adjacent to the supporting focus, and soon becomes diffuse throughout the apex. The type of reaction found was the same in gray or fat marrow and in red marrow. The process begins as a dilatation of vascular spaces, followed by the appearance of young fibrous connective tissue and infiltration by plasma cells, macrophages and lymphocytes. Osteogenesis had occurred in several cases, in areas surrounding the focus of suppuration and bone absorption. Different stages of this type of reaction are shown in Figs. 11, 14 and 18. This type of reaction bears no histological resemblance to the hyperplasia of bone marrow occurring in response to systemic infections or in certain diseases of the hematopoietic system, but is an inflammatory reaction which has taken place in bone marrow adjacent to a walled-in focus of suppuration or coalescent osteitis. The destructive process in the focus has destroyed cell partitions and sometimes encroached on marrow spaces, but the reactive process within the marrow has been adequate to prevent extension in that

direction and the suppuration has progressed along lines of lesser resistance, usually along vascular channels (without evidence of a thrombosis), through the cortex to the dural surfaces.

This process in the bone marrow is not a true acute osteomyelitis, in that it is not characterized by a diffuse destructive process within the bone marrow. It is a reactive, proliferative process, resulting in fibrosis, and, in some areas, sclerosis, which provides a strong line of defense against invasion of the marrow spaces by suppuration in adjacent areas of the bone.[1]

Acute osteomyelitis of the petrous pyramid was found in only one case of the series (No. 7) and in that instance it was a bilateral diffuse process, apparently of short duration and secondary to the bilateral labyrinthitis. The meningeal infection and labyrinthitis evidently followed the rupture of the localized abscess in the right pyramid. Also, one case of acute osteomyelitis of the squamous part of the temporal bone was included in the twenty-eight cases of meningitis.

BACTERIOLOGY

The infecting organism in this series of eleven cases of petrous pyramid suppuration was hemolytic streptococcus in seven, pneumococcus type III in three, and pneumococcus type I in one case.

The duration of the disease varied to about the same extent in the pneumococcus infections as in the streptococcus group. In both groups were instances where bone absorption was rapid, with erosion of the inner table and meningeal infection within two weeks, while in both groups also were instances where suppuration remained localized in the petrosa or in the extradural space for eight to eleven weeks with little subjective symptoms except the complaint of pain.

In every instance in which there was a febrile or septic course in the premeningitic stages, or a bacteremia, or where a sinus thrombosis was found, the organism was a hemolytic streptococcus.

THROMBOSIS OF VENOUS SINUSES

Among the clinical cases treated was one (case 10) which ran a febrile course, had a jugular block and apical empyema, one (case 11) with apical suppuration, bacteremia, and later jugular thrombosis, one case (successfully treated but not included in above report) with signs of early cavernous sinus thrombosis, and one fatal case, followed in consultation several years ago (not reported) with symptoms of petrous pyramid involvement, jugular thrombosis and retropharyngeal abscess, in which the petrosa was not explored.

The anatomical relationship of the venous sinuses to the petrous pyramid explains the extension of suppuration directly to the carotid sinus, the cavernous sinus, the superior petrosal, or more probably the inferior petrosal sinus, or to the jugular bulb. In such cases a lateral sinus block (as shown by the Tobey Ayer test) may be absent unless or until the jugular bulb or lateral sinus is involved.

It is evident from the anatomico-pathological findings as well as from the clinical experiences outlined above, that in those cases of sinus thrombosis which develop from a petrous pyramid suppuration, the drainage of the suppurating focus in the bone is the essential part of the surgical treatment, and unless this is accomplished, ligation of the jugular and removal of the clot may not prevent a meningeal extension.

PNEUMATIZATION

Studies that have been made on pneumatization of the pyramid, both by x-ray and by anatomical investigation, indicate that some degree of pneumatization of the apex is to be found in ten to fifteen per cent, while the posterior cell areas, medial to the arcuate eminence are pneumatized in a much higher percentage.

Pneumatization usually extends medially from the mastoid cells to the posterior cell areas, and tends to develop along certain tracts, namely, along the postsuperior angle, above the superior canal, along the subarcuate vessels, behind the posterior canal and aqueductus endolymphaticus, or along the retrofacial cell group and above the jugular bulb. In cases with a pneumatized apex the cell development may have extended from these posterior cell areas, either above or below the internal meatus and labyrinth, or it may develop from the middle ear or eustachian tube, without having any connection with the mastoid.[2]

In the cases reported here there are examples of pneumatization along all of the commonly found cell tracts. Of particular importance from a surgical point of view are the two examples (cases 2 and 5) in which the cells in the petrosa have developed directly from the middle ear and attic, respectively, and have no connection with the mastoid cells.

OPERATION

It is evident from the histopathological findings that no single operative procedure is suitable for all cases of petrositis. It is necessary to adopt a systematic approach that will avoid overlooking any localized areas of disease.

A discussion of surgical methods is not intended in this report, but certain essential considerations which are illustrated by the cases described, may be summarized.

The complete exenteration of the mastoid cells is the first essential, including careful search for tracts that might lead into the petrosa, and exposure of dura of the middle and posterior fossa. This procedure alone has undoubtedly been successful in many cases of suppuration around the labyrinth.

When the labyrinth is still functioning, and no fistulous tract is found, the dura must be elevated beyond the arcuate eminence in the middle fossa, in order not to overlook a localized focus of suppuration extending through the cortex in that area. (See cases 5, 6 and 7.)

These procedures may fail to reach suppuration in the apex, and both the histopathological findings and the clinical experience reported here indicate that the apical abscess is most frequently not accessible from the mastoid.

The presence of a fistula opening into the middle ear in such cases may allow a spontaneous recovery to take place, or when operation is indicated, it may act as a guide to the focus in the apex. However, in the cases reported here, there has been only one case (2) out of six with any sort of a fistula to the middle ear. The lesion in the apex has in our series almost always been a "closed" abscess.

The surgical approach to the apex which was used by the author in cases 9, 10 and 11, and one additional case, was a combination of the techniques recommended by Eagleton,[3] Ramadier,[4] and Kopetzky and Almour.[5] The exposure afforded by the radical operation was enlarged by removal of the processus epitympanicus, part of the zygomatic root, the anterior canal wall and the tensor tympani muscle. The apex was entered from in front of the cochlea by means of a eustachian tube curette. The apex was entered and the abscess opened without exposing the carotid in one case, while in three the carotid canal was first opened widely. The opening of the carotid canal appeared to provide more adequate drainage of the abscess in the apex. The function of the labyrinth was preserved in all four cases. There was a moderate hearing impairment for low tones, but the whispered voice can still be heard in three cases, while the fourth has not been tested. Although drainage was profuse and persisted for many weeks in every case, the aftercare presented no special difficulty.

Certain other extra-petrosal methods of surgical approach might be suitable for the case in which there is a large erosion of the intracranial surfaces of the pyramid, or a break through the inferior sur-

face[6] into the neck; however, there can be no question in the desirability of securing drainage from a suppurating focus in the pyramid before the intracranial extension has taken place, and therefore a safe method of opening the apex is a necessity. Although the intrapetrosal approach from the middle ear is not without some risk of hemorrhage,[7, 8] it provides the most practical method of draining the closed abscess of the apex.

SIGNS AND SYMPTOMS

Palsy of the abducens nerve occurred in three out of six cases of empyema of the petrous apex. In one of the remaining three (case 1) the palsy appeared after a diffuse meningitis had been present for several days.

Two cases of abscess in the apex (9 and 10) exhibited a positive Babinski's reflex (plantar extension) on the opposite side, before the appearance of the sixth nerve palsy and before there were changes in the spinal fluid. This sign was also observed in one case of temporal lobe abscess, otitic in origin, several weeks before the appearance of other localizing signs or spinal fluid changes.

It appears therefore, in order to explain the pyramidal tract involvement, that a local meningeal inflammatory reaction must have been present in the middle fossa in these cases, before changes were evident in the spinal fluid.

Plantar extension on the opposite side has apparently not been recognized as an early finding in petrous pyramid suppuration, but in the three cases mentioned here, it was an early and reliable indication that a complication was present.

A review of the symptomatology in this series of cases shows that the one complaint or symptom common to all cases was pain. The history obtained in those cases in which the otitis had remained unrecognized until meningitis had developed (6 and 7) revealed that pain had been present, referred to the region of the ear, and had been overlooked. A persistent aural discharge of purulent character was common, but in two cases the discharge had ceased and in two it had never appeared. A high fever was present throughout the disease in some cases, and in others, after the initial stages of the otitis, fever was absent until meningitis developed. Vomiting occurred in most cases before the onset of a diffuse meningitis, and was an important indication of a complication. The presence of pain or headache was the most constant and reliable symptom, and the location of the pain, at least during the developing stage of the complication, was a valuable indication as to the site of the pathology.

ROENTGENOGRAMS

The roentgenograms have been an important aid in the diagnosis, particularly when the lesion has been located in the apical part of the pyramid. The occiput-down views, Stenver's position, and the axial views are used routinely, and serve to show the pyramid from different angles (the technique has been well described by Lysholm[9]). In our experience the axial views have been most useful in demonstrating an asymmetry of the petrous apex, particularly during the intrapetrosal stage of the infection.

Evidence of pneumatization in the healthy apex, or in both apices is important, since suppuration in the apex occurs most frequently in pneumatic spaces. Increased density on the diseased side may be seen (case 10), or, in late cases, a decreased density and loss of trabecular markings may be shown (case 9). A general increase in density on the diseased side may be expected in unilateral osteomyelitis of the apex, or in the presence of a reactive fibrosis (as in case 6).

The roentgenogram usually furnishes valuable confirmatory evidence regarding the presence or absence of disease of the apex, and may show up gross erosions of the cortex in late cases. It does not provide any reliable indication as to when or where the intracranial extension is likely to take place.

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II

THE WIDTH OF THE BASILAR MEMBRANE IN MAN*

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PRINCETON, N. J.

Practically all theories of hearing are based upon particular assumptions regarding the physical characteristics of the basilar membrane. These assumptions are most definite for the place theories, which account for pitch discrimination by the differential action of regions of the membrane. The resonance type of place theory, as maintained by Helmholtz and his followers, considers the physical properties of the membrane to vary systematically throughout its length, so that separate fibers or bands respond selectively to tones of different frequency.

The physical features that have received most consideration in relation to the differential response of the membrane are those that determine the natural frequencies of stretched strings, namely, the width of the membrane, and the mass, tension and loading of its transverse fibers. A further dimension that obviously is involved is the length of the membrane from base to apex.

Only this last dimension is accurately known. Recent measurements made in this laboratory by Hardy[1] have shown that the membrane in man has an average length of 31.5 mm., and a range of variation of 25.3 to 35.5 mm. Tension has been measured at one position in the basal turn by Berendes,[2] but its variation throughout the cochlea has never been ascertained. It is often assumed that it varies with the size of the spiral ligament, which decreases from base to apex. The mass of the membrane and the structures which it bears also varies from base to apex, and it has been supposed by some that there is differential loading of the membrane by fluid columns; but of these features we have no quantitative knowledge.

This paper is concerned with the width of the basilar membrane. In man, this dimension has been measured in only eight or nine cochleas, so far as the writer is aware, and in most of these in but a few roughly indicated positions.

The first observations were made in 1863 by Hensen[3] on a newborn child. Measurements at seven places from base to apex

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indicated a total range of 41 to 495 micra (0.041 to 0.095 mm.). These data were used by Helmholtz when he revised his theory, in line with Hensen's suggestions, to make the basilar membrane the differentiating mechanism in the cochlea.

Retzius[4] in 1884 reported measurements on an adult, taken at three positions. The widths observed were 210, 340 and 360 micra in basal, middle and apical turns, respectively.

The best observations are those of Keith,[5] who in 1918 reported measurements on four specimens, one at five and the others at six positions, with estimates of the locations of these positions along the membrane. The measurements on one specimen, prepared by Fraser and Dickie, ranged from 176 micra at a position about 7 mm. from the basal end to 400 micra at a position about 33 mm. from the end. The average results from the other three specimens were 160 micra near the base and 520 micra near the apex.

Held[6] in 1926 obtained values of 176, 304 and 480 micra in basal, middle and apical turns, respectively, in a 41-year-old man. A second set of measurements was given as 104, 336 and 504 micra, for the same locations. A measurement at the extreme basal end in another adult specimen yielded a value of 80 micra.

Similar observations have been made on several other species of mammals. Hensen remarked that the basal end of the membrane in the ox was more "pointed" than in man, but gave no measurements. Retzius reported a few figures for each of six rabbits and eight cats. Kolmer[7, 8] in 1907 and 1909 studied the membrane in a number of species, including several domestic animals and primates, but his measurements were limited usually to one or two specimens of each species. Wada,[9] in his developmental study of the ear of the rat, gave measurements at four positions for a number of animals of different ages from 1 to 546 days; of these perhaps thirteen might be regarded as having reached a mature stage of development of the basilar membrane.

All of the foregoing studies indicated a progressive increase in the width of the basilar membrane from base to apex.

The only study that has employed a sufficient number of animals to show the range of individual variations within a species is that of Guild[10] on the guinea pig. Measurements were made at nine positions on each of 35 cochleas, with graphic reconstruction of three of these for a determination of the general positions of the measurements. These data showed, contrary to the prevailing conception, that the width of the basilar membrane varies greatly in its rate of

increase from the basal end upward. Moreover, it was found to attain its greatest width nearly a full turn from the apical end, and thereafter to narrow rapidly. The difference between maximum and minimum widths was a little over three-fold on the average.

METHOD AND PROCEDURE

The present study was made on human cochleas in serial section, with every fifth (sometimes every tenth) section mounted. The thickness of section was 24 micra in all but two series. Detailed measurements were made on 25 cochleas, of which 20 were sectioned in the vertical plane of the petrous pyramid, and five in the horizontal plane.* Hearing, as determined by audiometer tests before death, was normal in ten of the vertical and in the five horizontal series; the remaining ten vertical series included eight cases of high-tone deafness and two cases of otosclerosis.

In addition to the above material, three special series of sections and two gross specimens were used for further study of the basal end of the membrane, as described below.

Measurements were made at 17 to 19 positions, depending upon the number of cochlear turns. These included five or six radial positions and twelve or thirteen oblique positions.† These positions were determined for each cochlea on the basis of a graphic reconstruction by the method described by Guild.[11]

The graphic reconstruction method yields an ortho-projection to scale of the line of junctions of the pillar cells of the organ of Corti. The procedure for vertical sections is outlined here; that for horizontal sections follows the same principles.

Those sections are identified in which the junctions of the pillar cells are cut tangentially, which occurs once in each half-turn. The points thus found are plotted on cross-section paper along an axis perpendicular to the plane of section at distances determined by order and thickness of sections. These points are connected by semicircles to produce a figure approximating a spiral form, which represents the middle portion of the cochlea. Plotting the upper apical and lower basal portions requires further measurements. The

*The vertical plane of the petrous pyramid is nearly parallel to the plane of the superior semicircular canal; both it and the horizontal plane are parallel to the modiolar axis of the cochlea.

†Radial positions are those in which the plane of section passes through the center of curvature of the portion of basilar membrane concerned; this happens at but one place in each half-turn. (See Fig. 1.) All other positions are oblique, up to the point where the membrane is cut only on a tangent.

apical end is located, and with a screw-micrometer ocular its distance is measured from the lower apical turn in the same section. This distance, transferred to the scale of the graph, determines the arc drawn from the apical portion already plotted. Plotting the lower basal turn takes two steps. First, the region of greatest "bulge" is found as the midpoint of the sections in which the inner and outer pillar cells are cut radially and thus are seen entire. The distance from the greatest "bulge" to the upper basal turn is measured, and the point thus determined is connected with the upper basal junction by an arc whose center is located on the perpendicular axis. The second step is less precise, since the lower basal end lies in sections which contain no other cochlear turns, and a measurement of its location lateral to the perpendicular axis cannot be made. Therefore, this location is estimated on the basis of a study of horizontal sections, in which the form of the basal end is more clearly shown.

The type of projection obtained by this procedure may be judged from the inner border of the band shown in Fig. 4.

In the measurements of width of the basilar membrane the end points were taken as the attachment to the spiral ligament on the one hand and the expansion into the fibrous tissue under the limbus on the other. Since at each end the basilar membrane thickens gradually, the readings unavoidably involved a considerable element of judgment. The essential basis of the judgment was the apparent mobility of the membrane.

In radial positions, such as those shown as *i*—*j* and *k*—*l* in Fig. 1, the widths were obtained directly from the readings of the calibrated screw-micrometer ocular. For all other regions, in which the membrane was cut obliquely, further calculations were required. For example, the distance *c*—*b* in Fig. 1 was found by measuring *a*—*b*, and then drawing the arc *a*—*c* from the center *o'* so as to intersect with an extension of the line *o'*—*b*.

The accuracy of this method for oblique positions depends upon the definiteness of the end points and upon the accuracy of the graphic projection. The end points can be judged with sufficient exactness if care is taken that the section is not too near the tangential position (*b*). An error of projection arises insofar as a figure made up of a series of semicircles departs from the true form of the cochlea. On the assumption that the cochlea is a smooth spiral, this error is such that a measurement in the upper part of each half-turn (as *d*—*e*) is too small, and a measurement in the lower part of each half-turn (as *a*—*b*) is too large. Hence, following this assumption, the adjacent oblique values, like *c*—*b* and *d*—*f*, were averaged and the

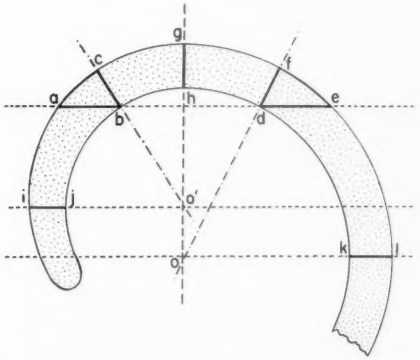

Fig. 1. Diagram to illustrate the determination of widths of the basilar membrane, particularly at oblique positions. The plane of section is vertical, and is indicated at three levels by short-dashed lines. Radial positions are shown at *i*—*j* and *k*—*l*, and oblique positions at *a*—*b* and *d*—*e*. Radial distances are also shown as *c*—*b*, *g*—*h* and *d*—*f*; these distances can not be directly measured in these sections, but can be found by geometrical construction (see text). The section which passes through the point *h* is a tangential section. The dashed line *g*—*o* is the perpendicular axis.

result taken as the tangential distance *g*—*h*. In these computations, no effort was made to take account of inequalities in the tangential distances, *b*—*h* and *h*—*d*. Also, no correction was made for the fact that the projection neglects the height of the spiral and thus reduces all linear distances by about one-third of one per cent.

RESULTS

Typical results of the measurements are shown in Figs. 2 and 3. Fig. 2 presents curves for the five shortest and Fig. 3 for the five longest among the group of cochleas from persons with normal hearing and sectioned in the vertical plane. In these figures, widths of the basilar membrane in micra are given on the ordinate, at positions indicated along the abscissa as distance in mm. from the basal end. The filled erect triangles designate measurements at radial positions, the open erect triangles measurements at oblique positions, and the open inverted triangles the average of two adjacent oblique measurements.

The curves do not always indicate the total length of the basilar membrane, because often the apical end of the cochlea lay at or near a tangential position (as *h*, Fig. 1), where measurements are difficult.

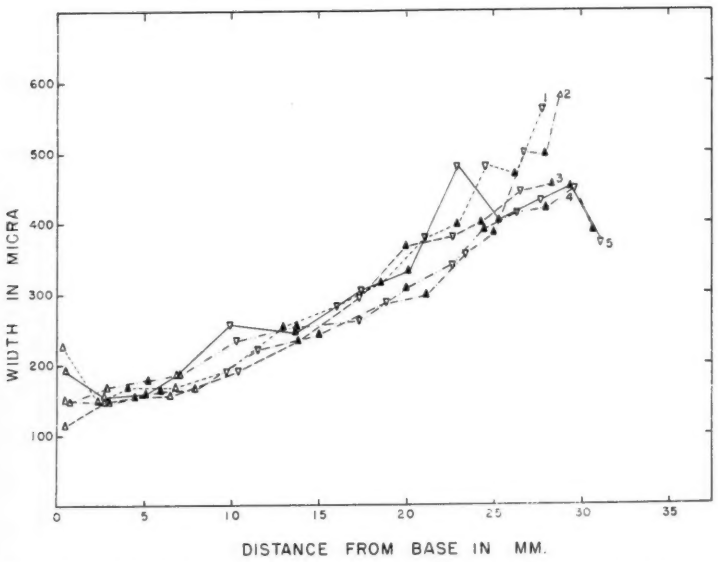

Fig. 2. Width of the basilar membrane in five normal cochleas of the "short" type, as measured at various positions from base to apex.

The curves show that the basilar membrane increases in width from the base upward, at first slowly, then more rapidly. In many cases a maximum is reached in the apical region, after which there is a rapid decline. See, for example, the curves marked 4 and 5 in Fig. 2, and 6, 7, 8, and 10 in Fig. 3. It will be noted that the decrease in width at the apical end is somewhat more marked for the longer cochleas. As mentioned above, Guild found the basilar membrane of the guinea pig likewise to attain its greatest width a half-turn or more before the apical end, and then to decrease rapidly.

In general, the basilar membranes of the shorter type, as shown in Fig. 2, present a slightly steeper rise throughout the upper half of their lengths, so that in the apical region their maximum widths are in the same range as those of the longer type.

The measurements at the extreme basal end show large variations. Apart from these, the values for the different cochleas are in good agreement in the basal turn. But as the apical end is ap-

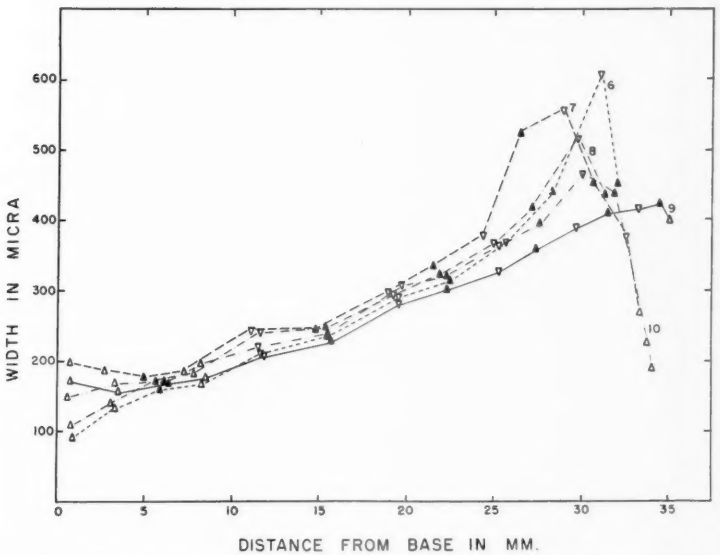

Fig. 3. Width of the basilar membrane in five normal cochleas of the "long" type.

proached, the variations become more and more pronounced. Some of the basilar membranes, like Nos. 1 and 2 in Fig. 2, increase in width up to the last position of measurement, while others reach a maximum and decline at various rates

The variations in the measurements near the basal end were due mainly to the fact that in this region the membrane departs from a spiral course. Between 3 and 4 mm. from the basal end it quickly twists from approximately a vertical plane to the horizontal, and then bends around in a ventro-medial direction to form a hook, the end of which lies at the lower edge of the round window. The form of this vestibular portion of the cochlea varies considerably in different persons, and thus the sections have various orientations relative to the plane of the membrane. The graphic reconstruction of this end is inexact, and no proper treatment of the measurements is possible.

A preliminary study of this part of the cochlea was made in gross specimens in which the bone was cut away to expose the lower basal turn. Examination showed the membrane to be fairly uniform in its rate of taper, and to end bluntly.

Two series were available that were sectioned in the frontal plane, that is, perpendicular to the modiolar axis. These sections were favorable for a study of the last 1.5 mm. of the basal "hook." They showed a gradual taper to a lower value of about 100 micra near the basal end.

A more exact study of the basal end was made in one cochlea which was sectioned especially for the purpose. The block was observed with a microscope during the process of sectioning, and reoriented as often as necessary to maintain a radial position with respect to the membrane. Measurements were made on this series at short intervals throughout the last 6.5 mm. of the membrane. The results agreed well with those plotted in Figs. 2 and 3 for cochleas 3, 6, and 8. Below 1 mm. from the base the width decreased rather rapidly to a value of 80 micra at a point 0.24 mm. from the end. This measurement at the basal tip thus agrees with that reported by Held, as cited above.

The measurements of the five horizontal series of normal cochleas and also those of the ten vertical series of cochleas from persons with defective hearing were not significantly different from those shown in Figs. 2 and 3.

An examination of the results revealed no differences that could be related to age, sex, or race (white vs. American negro).

The group of 25 cochleas included six pairs; a comparison of right and left cochleas showed that three of the pairs were similar in form, and the remaining three rather different in form at the apical end.

On account of the large variations of form it was not considered advisable to average the results of the entire group. However, in Fig. 4 is given a somewhat idealized representation of the basilar membrane, on a scale 10 times natural size, in a cochlea of rather more than average length. The basal tip in this figure was drawn from the series of specially oriented sections, the middle portion follows the average of the group of 25 cochleas, and the apical end represents a particular case that was selected as typical of cochleas of the long type.

Since many theories of hearing have regarded the basilar membrane as a factor in the differential tuning of the cochlea, the range of variation of this dimension needs special attention. As shown above, the maximum width for a given cochlea is found sometimes near the apical tip, but more often some 3 to 6 mm. before this end is reached. The largest maximum observed among the 25 ears was 651 micra, in

Fig. 4. Sketch of the basilar membrane, to show the variations of width throughout its course. Scale, 10 times natural size.

the middle of the lower apical turn, 29 mm. from the base, for a membrane whose total length was 31.4 mm. The smallest maximum observed was 423 micra, in the upper apical turn, 34.5 mm. from the base, in a membrane 35.0 mm. long. If we accept 80 micra as a probable value for the minimum width for both these cases, we have a variation of a little over eight-fold in the first instance, and somewhat over five-fold in the second. Both these ears were normal in function so far as indicated by audiometer tests. And though no specific data are available regarding the keenness of pitch discrimination, these persons exhibited no obvious differences in the ability to understand speech. It is evident that any theory that postulates a differentiating role for the width of the basilar membrane must allow for a wide range of normal variation at the apical end. The results certainly suggest that the operation of the factor of width in the apical region can hardly be specific.

The average value of the maximum among the 25 cochleas is 498 micra, which in relation to 80 micra indicates a six and one-

fourth-fold variation. This value is about midway between the older figure of twelve-fold as given by Hensen and featured in the theorizing of Helmholtz, and the three-fold variation indicated by later observations and accepted in most of the recent discussions.

SUMMARY

The width of the basilar membrane was measured in 25 human cochleas at a number of positions from base to apex. By the graphic reconstruction method, these positions were determined for each cochlea in terms of the distance from the basal end.

The measurements for the different ears were in good agreement in the basal part of the cochlea, but showed wide variations in the apical part. Most of the basilar membranes, and particularly those of more than average length, attained a maximum width at a point about a half-turn before the apical end, and then decreased rapidly.

The average value of the maximum points measured was 498 micra, which is about six and one-fourth times the minimum width of 80 micra as measured in a special series of sections.

No significant relation was found between the form of the basilar membrane and age, sex, or race, or the condition of hearing as shown by audiometer tests made before death.

It is pointed out that a theory of hearing that postulates a differentiating röle for the width of the basilar membrane must allow for a wide range of normal variation in the apical region.

ENO HALL.

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III

DEEP INFECTIONS OF THE NECK*

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NEWARK

My interest in deep infections of the neck was aroused many years ago following an unfortunate experience, and I am deeply indebted to Dr. Mosher for the stimulus given me to approach these infections; this was in 1929, and I strongly urge those who have not read his Presidential Address before the Academy of Ophthalmology and Otolaryngology to do so.

Much has been written since 1929, at which time Dr. Mosher[1] described his submaxillary fossa approach to deep pus in the neck, and it would be superfluous to attempt to add to his incomparable description of the fascial planes in the neck, along with those of Barnhill[2] and Furstenburg,[3] other than to emphasize certain points. Laryngologists, particularly those who are doing esophagoscopy, should not hesitate to operate early in this type of infection.

This paper is based mostly on infections of the buccopharyngeal fascia which have come under my care.

In one of our hospitals we see a large number of infections of the submaxillary region, and these are usually from infections in and around the teeth and tonsils. In another hospital we see a fairly large number of deep infections, mostly infected glands following scarlet fever and measles. It would be taking up too much valuable time to speak of each patient individually.

The material included in this paper is based on sixteen cases of deep infection, particularly low in the neck, with three deaths.

The various locations of these infections are submaxillary, pharyngomaxillary, carotid sheath and buccopharyngeal spaces.

In this series there are nine female and seven male patients, the age of the females ranging from 18 to 72 years, and of the males from 42 years to 63 years.

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*Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, January 7, 1938.

ETIOLOGY

An accurate history of the patient is of the utmost importance, for only by obtaining this can we expect to find the portal of entry of the infection. With this information we are able to anticipate its course.

We have in this series one case in which the teeth were the cause of an abscess in the submaxillary fossæ; the tonsils were the cause of infection in three cases in the pharyngomaxillary space, and three in the carotid sheath; while nine were infections in the buccopharyngeal space from the hypopharynx. Treatment was by external operation in all but one case, with thirteen recoveries and three deaths. All three deaths were from infections in the buccopharyngeal space.

Complications: Two patients had abductor paralysis. In one it was bilateral, necessitating a tracheotomy.

The predominating organism was hemolytic streptococcus. In the last case we found fusiform bacilli and spirillum of Vincents.

APPLIED ANATOMY

The pharyngomaxillary fossæ is bounded above by the base of the skull, internally by the superior constrictor muscle of the pharynx, externally by the internal pterygoid muscle and ramus of the mandible, and the inner prolongation of the parotid gland. Pus in this area can drain down along the carotid sheath, or, as Mosher has so termed it, "the Lincoln highway of the neck."

Paths of Infection for Abscesses of the Neck: Abscesses of the neck arise chiefly from cervical lymph glands in which are retained infective agents from a regional inflammatory process. The further extension of the pus follows along certain definite channels, i. e., the connective tissue planes of the cervical fascia, and there are four usual connective paths along which an inflammatory process may extend:

1. Superficial.
2. Pretracheal.
3. Buccopharyngeal.
4. Prevertebral.

The part that I am trying to stress in this paper is the buccopharyngeal. The two layers of pretracheal fascia which surround the thyroid gland unite at the posterior surface of the thyroid gland and pass across behind the pharynx and the esophagus, and are known

as the buccopharyngeal fascia. This fascial plane communicates directly with the posterior mediastinum.

Mosher and Jackson have called attention to the weak point in the esophagus where the inferior constrictor fibers split, which permits a herniation, and is the weakest spot just behind the cricoid cartilage. It is in this area that most of the infections, either by trauma or perforation, cause the rapid spreading of infection upwards and downwards in the buccopharyngeal space. Unless we have the courage to drain this area immediately, death will usually follow, although we do read occasionally of cases in which drainage has been made successfully from within.

SURGICAL APPROACH

Deep infection of the neck calls for adequate drainage by means of a long incision, and unless we make it so we are apt to find ourselves working in a funnel.

Using Mosher's "T" incision in his submaxillary approach, one may reach pus in the pharyngomaxillary fossæ, side of tongue, floor of mouth and region of tonsil, and by extending the vertical line of the incision downward along the anterior border of the sterno-cleido mastoid muscle, infection may also be reached in the carotid sheath and buccopharyngeal fascia.

In approaching the buccopharyngeal fascia, the vessels are retracted outward while the trachea and thyroid glands are retracted inward; remembering that the two layers of pretracheal fascia which surround the thyroid gland unite at the posterior surface of the gland and pass across behind the pharynx and esophagus, as the buccopharyngeal fascia. This space is entered either by finger or hemostat. With infections in this area we are likely to find the fascia rather dense.

If it becomes necessary to do a cervical mediastinotomy, after the procedure of Furstenburg, it is well to remember that the esophagus is in close relationship with the pleura high up on the left side, whereas, on the right side, the esophagus and pleura come in contact at a lower level. Therefore, we run less risk of injury to the pleura on the right side.

I wish to call attention to the possibility of trauma to one or both recurrent laryngeal nerves from deep infection in this area. The late Edward F. Ziegelman[5], in his article on laryngeal nerves said, "Most of these lesions are due to trauma of some kind incurred during surgical procedures on the thyroid gland. Judd, New and Mann demonstrated experimentally the effect of trauma on this nerve. Their

conclusions seem to prove that stretching does not impair the function of the nerve. Ligation and severing, they found, result in permanent paralysis, while pinching causes temporary paralysis of the corresponding portion of the cord."

SUMMARY

1. Adequate drainage is essential for pus deep in the neck.

2. With a history of chills and a negative blood culture, adequate drainage may be sufficient without ligation of neighboring veins.

3. Abductor paralysis should not be overlooked as a result of deep infection low in the neck.

4. We should not be misled by a falling temperature, with a general feeling of well-being and think the patient is recovering. With the tender spot on the side of the neck still persisting, it is but the calm before the storm.

5. High leucocyte count from 16,000 to 26,000 is a significant point.

6. Cervical mediastinotomy may be required in addition to free drainage of the neck.

7. Perforation of the hypopharynx, instrumental or by foreign object, with or without marked emphysema, but with a persistent tender spot over the posterior border of the thyroid gland, with a high leucocyte count—have the courage to go in and drain that area.

8. An accurate history to enable one to visualize port of entry of possible infection, and the anatomical planes which the infection may follow is essential.

9. Lateral roentgenograms of the neck usually show considerable displacement of the trachea anteriorly. This especially applies to infections from the hypopharynx.

CONCLUSIONS

Delay in instituting early and courageous treatment in deep infections of the neck, particularly in the lower half, may prove disastrous.

I believe the persistent tender spot over the posterior border of the thyroid gland is a diagnostic point, and recommend immediate drainage.

24 COMMERCE ST.

Case No.	Name	Sex	Age Yrs.	Etiology	Fossa Submaxillary	Pharyngo-maxillary Space	Bucco-pharyngeal
1.	F. D.	M	60	Teeth	Abscess	.....	.....
2.	A. N.	M	45	Tonsils	.....	Abscess	.....
3.	A. H.	M	53	Hypopharynx esophagus	.....	.....	Abscess
4.	M. M.	M	52	Esophagus bone	.....	.....	Abscess
5.	C. C.	F	50	Esophagus bone	.....	.....	Abscess
6.	H. S.	F	41	Esophagus bone	.....	.....	Abscess
7.	M. E.	F	18	Tonsils	.....	Abscess	.....
8.	M. B.	F	49	Hypopharynx bone Esophagus	.....	.....	Abscess
9.	E. W.	F	51	Tonsils otitis	.....	.....	.....
10.	J. K.	M	47	Hypopharynx bone	.....	.....	Abscess
11.	M. S.	F	29	Tonsils	.....	.....	.....
12.	M. R.	F	72	Hypopharynx bone	.....	.....	Abscess
13.	J. F.	M	42	Hypopharynx bone	.....	.....	Abscess
14.	P. F.	F	53	Tonsils	.....	Abscess	.....
15.	H. J.	F	19	Tonsils	.....	.....	.....
16.	N. S.	M	63	Hypopharynx	.....	.....	Abscess

Carotid Sheath	Treatment	Complications	Results	Bacteriology	Case No.
.....	External drainage	.....	Recovered	.....	1
.....	External drainage	.....	Recovered	Non-hemolytic strep.	2
.....	Gastrostomy	Mediastinitis P-M. Ca. stomach	Died	.....	3
.....	Ex. drainage. Cervical mediastinotomy	Abductor paralysis	Recovered	Hemolytic strep.	4
.....	Ex. drainage. Cervical mediastinotomy	.....	Recovered	Non-hemolytic strep. and staph.	5
.....	Ex. drainage. Cervical mediastinotomy	.....	Recovered	Hemolytic strep.	6
.....	External drainage	.....	Recovered	Non-hemolytic strep.	7
.....	Ex. drainage. Cervical mediastinotomy	.....	Died	Hemolytic strep.	8
Abscess	External drainage	.....	Recovered	Non-hemolytic strep. and staph.	9
.....	Ex. drainage. Cervical mediastinotomy	Bilateral abductor. Tracheotomy	Recovered	Non-hemolytic strep. and staph.	10
Abscess	External drainage	.....	Recovered	Hemolytic strep.	11
.....	External drainage	.....	Recovered	Non-hemolytic strep. and staph.	12
.....	External drainage	.....	Recovered	Diphtheroids. Staph. albus.	13
.....	External drainage	.....	Recovered	Hemolytic strep.	14
Abscess Chills prior to operation Blood culture, neg.	External drainage	.....	Recovered	Hemolytic strep.	15
.....	Ex. drainage. Cerv. medias'y. Gastrostomy	.....	Died	Vincents and staph.	16

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IV

MÉNIÈRE'S DISEASE AS AN INDICATOR OF DISTURBANCES IN THE WATER METABOLISM, CAPILLARY FUNCTION, AND BODY CONDITION

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AND

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Furstenberg, Lashmet and Lathrop[1] have recorded some excellent investigations of the influence of the water and salt metabolism upon Ménière's syndrome on the basis of which they consider it justifiable to establish that, in case of this affection, there is a question of retention of sodium and not, as we assert, of retention of water. Furstenberg, and after him Madelaine R. Brown,[2] have subsequently subjected such patients to a treatment consisting of a diet poor in sodium chloride instead of a diet at the same time poor in water, as suggested by us. Földes in his "New Approach to Dietetic Therapy"[3] recommends a diet with reduced content of fat, carbohydrates and water (one liter) but with no restriction of salt, and with plenty of proteins.

Although our works have roused a certain interest, only a small and casual selection of them has been quoted. This probably is the reason why very essential facts, such as the hearing and general condition of the patient, have escaped the attention of these authors. Furstenberg's experiments, if correctly interpreted, as will be seen later, in reality perfectly agree with our observations. We also shall endeavor to explain why Furstenberg as well as Földes have been able to obtain similar results with their apparently very different treatments.

We thought it might be of interest in a brief synopsis to account for the result of our works, with the inclusion of our latest corrections and additions, the details of which will be found in our previous publications.

I. *The Clinical Picture of Ménière's Disease.*—Primarily we define Ménière's disease as an ear disease with periodically varying acoustic and vestibular symptoms, which do not present any special etiology

(such as for instance middle ear suppuration, syphilis, brain tumor, trauma, etc.).

On the basis of many hundred observations, for about 175 of which we have previously[4, 5] accounted in detail, it may be said that this affection is of very frequent occurrence, but that its nature very often is not recognized. It is of equally frequent occurrence in the two sexes, and we have met patients of all age classes with the exception of the first years of life.

In the majority of cases the otoscopy reveals the tympanic membrane to be fibrous or atrophic, and very often retracted.

Tubal stenosis is of frequent occurrence, particularly during the bad periods of the disease.

The hearing is nearly always reduced, even though this sometimes is not detected without a detailed functional test. Several of the patients whose hearing originally has been normal, subsequently become more or less deaf. In most cases both ears are affected though in a different degree. Almost always the hearing is most severely impaired on the side where the otoscopic changes are most pronounced. We always employ tuning-forks and the monochord which, in the hands of a skilled examiner, yield very reliable results. In our opinion some audiometers have, for instance, the drawback that they do not permit of accurate determinations in the highest and the two lowest octaves, where the reduction of audition is most marked. Practically there is always a question of a distinct sound conducting affection with a typical elevation of the lower limit (bass deafness). In cases of pronounced deafness the upper limit is lowered also (treble deafness). This was formerly considered indicative of a loss of sound perception due to a secondary nerve degeneration. However, Dida Dederding in subsequent investigations[6, 7] determined that this lowering of the upper limit is only found when tested by air conduction, whereas, if tested by bone, it remains normal, showing that the perceptive organ must be intact. Numerous other cases of typical affection of sound conduction, which may be caused even by a mere plug of cerumen in the external meatus, present the same lowering of upper limit by air but not by bone conduction.

The results of the testing of bone conduction for a_1 also indicates an affection of the sound conduction. This likewise applies to those cases, where bone conduction, as compared to air conduction, is considerably shortened or even quite absent; for in agreement with Reitö[8], but in opposition to the commonly held conception, we cannot

PLUG OF WAX IN RIGHT EAR

Upper limit air c.	Upper limit bone c.	a_1 Air Cond. (normally 60″)	a_1 Bone Cond. (normally 20″)	Lower limit	Whisper
10.000 d. vbr.	17.000 d. vbr.	21″	18″	90 d. vbr.	1.50 m
15.000 d. vbr.	17.000 d. vbr.	60″	20″	16 d. vbr.	20.00 m

AFTER REMOVAL

15.000 d. vbr.	17.000 d. vbr.	60″	20″	16 d. vbr.	20.00 m
15.000 d. vbr.	17.000 d. vbr.	60″	20″	16 d. vbr.	20.00 m

acknowledge a comparatively shortened bone conduction as a sign of an affection of the sound perception, as we have substantiated in detail in previous works[6, 7, 9].

The most characteristic feature of hearing is its frequently varying nature. It can change from one day to another (for instance from 0.50 to 10.00 m. whispered voice) or within some hours (for instance from 1.50 to 20.00 m. or from 0.30 at 9 o'clock a. m. to 20.00 m. at 5 o'clock p. m.) or even instantaneously, and in this case it is as a rule accompanied by an attack of giddiness.

The majority of patients complain of buzzing in the ears, which generally occurs in highly varying periods also. However, this symptom may be absent, or in some patients periodic, regardless of the degree of deafness. The buzzing may even be accompanied by an improvement in their hearing.

Autophony is by no means of rare occurrence. This also holds good of diplacusis.

A phenomenon which, beside the middle ear changes and the bass deafness, decidedly speaks against these patients suffering from a central affection, is the very common sensation of fulness or pressure in the ear. It is as though "there were water in the ear," which the patient tries to get rid of by turning a finger in the external meatus or pulling it. This sensation, which may amount to acute pain referred to the maxillary joint, is identical to the sensation experienced in a mountain-railway or an airplane, being particularly pronounced in case of tubal stenosis.

All the patients at least periodically present spontaneous nystagmus[10] the detection of which depends upon the frequency and thoroughness of the examination. In the majority of cases the

nystagmus is of a horizontal-rotary character. However, oblique and vertical or combined forms may also be observed. The nystagmus is about twice as often directed to the more affected ear, as to the other side. However, the hearing of patients with such "heterolateral" nystagmus is not, as a rule, inferior to that of patients with "homolateral" nystagmus. That the direction of the nystagmus is of secondary importance is moreover evidenced by the facts that numerous patients during one attack may present nystagmus in one direction and during another attack in the opposite direction, and that we have repeatedly witnessed alternations in the direction of the nystagmus during one and the same attack. Extremely rare are signs of a reduction of the vestibular function observed by rotation or caloric tests. Nystagmus due to the posture of the head or inhalation of amyl nitrite is of very frequent occurrence. It acquires great diagnostic importance in cases in which abnormalities of nystagmus are not otherwise detectable. Not infrequently a fistula symptom may be elicited by compression and aspiration in the external meatus, in isolated cases even by compression of the carotid, without its having anything to do with syphilis. During catheterization giddiness and nystagmus, which as a rule is homolateral, are very frequently observed.

Giddiness may occur in all possible forms and degrees, ranging from the slightest unsteadiness to the most violent attack of several days' duration. In the same patient it may vary extremely at different times. It may accompany congestion, perspiration, salivation and diarrhea. As was already mentioned, such an attack of giddiness may cause a reduction or an improvement of hearing (Lermoyez's "vertige qui fait entendre"). Both these phenomena are sometimes observed in one and the same patient.

However, these patients do not present ear symptoms only. The majority of them present numerous other morbid phenomena, varying in location and combination from case to case, though always with the same periodical alternations as those for the ear disease.

One of the phenomena, which almost never fails to appear, is headache[11]. It may be quite dominant and, in isolated cases, have the character of migraine. Most frequently it is homolateral with the deafness. During the bad periods moreover the patients sometimes suffer weakness of memory, depression and attacks of irritability, which may erroneously suggest encephalitis. Very slight changes in the pressure, number of cells and albumin content of the cerebrospinal fluid are quite common.

Disturbances of circulation are of very frequent occurrence, though not affections of the heart or of the large vessels, liver or kidneys. They are chiefly of a vasomotor nature deficiency. Particularly during the bad periods the skin is cold, pale or cyanotic. This is especially observed as acrocyanosis of the feet, hands, nose and ears. Besides, even in the middle of the warmest season, the patients often are shivery.

Subcutaneous infiltrations are very frequently observed, not only in women where they must be considered in a certain degree physiological, but also in men. In contradistinction to edema proper they are very tender and not plastic, finger pressure, for instance, leaving no marks. They are most marked in the dependent regions and in places, which are particularly exposed to cold, such as, for instance, the nape of the neck in males and the outward surface of the upper arm in females. They increase and become more tender during the bad periods[12].

On the mastoid process, particuarly on the side most severely affected with headache and deafness, tenderness is frequently observed, and sometimes palpable infiltration which extends along the nuchal line. This peculiar mastoidal tenderness has previously been described as belonging to "Bárány's syndrome."

Among other comparatively frequent concomitant phenomena ([4, 5, 13, 14]) may be mentioned vasomotor rhinitis, various gastrointestinal disturbances and rheumatism. The blood pressure is oftener low than high. It may be mentioned that Lian and Blondel[17] have described a "hypotonic syndrome" consisting of vertigo, fatigue, acrocyanosis and other vasomotor symptoms, scanty diureses and changing condition. In some cases we have seen Quincke's edema appearing simultaneously with Ménière's syndrome[15].

We shall now proceed to a discussion of a series of phenomena ([4, 5, 13]) which naturally tend to suggest a relation between Ménière's disease and disturbances in the water metabolism.

As was mentioned before, we rapidly became aware of the strikingly small diureses in these patients who do not otherwise present any signs of renal or cardiac insufficiency. However, such small diureses sometimes occur during the daytime only, whereas during rest in bed at night, there is a copious output (nycturia). The small diureses frequently coincide with the patients' periods of increased deafness, buzzing, headache and indisposition. Inversely, good periods are found to be initiated by increased diureses. Moreover, during the bad periods there is a tendency to increase, and during the good periods to loss of weight. Prior to menstruation diuresis

is likewise scanty, the symptoms often at the same time increasing. This is likewise the case after infections which also generally exert an exacerbating influence. Frequently a sudden increase of diuresis is observed following an attack of giddiness. Moreover, such an attack may be accompanied by a considerable loss of weight because of excessive salivation, vomiting and diarrhea. Sometimes, apparently paradoxically, the more violent such an attack has been, the greater is the relief subsequently experienced by the patient. Some patients when they feel worst actually long for such an attack, which thus seems to act as a natural means of regulation.

To many patients perspiration is difficult, and they feel strikingly relieved as soon as they begin to perspire after physical exercise. Also the feces may be scanty, at any rate many patients have a tendency to constipation which, at times, may suddenly change to diarrhea, especially during the attacks.

Many patients have themselves noticed that ingestion of much liquid does not agree with them. One reported that at the beginning of a dinner party she heard very well, but gradually became deaf as the last course approached, particularly if the first course had been broth. Therefore a good many patients have on their own accord reduced not only their consumption of liquid but also of salt.

Similar observations are already on record in the older literature without the connection being understood. Thus Sharp[16] reports the case of a deaf woman who used to drink 10 or 12 cups of tea a day. When she abstained from this "poison" her deafness disappeared. It recurred, however, when she resumed her tea drinking. Banting, who was not only very stout but also deaf, obtained by his famous treatment of obesity not only a loss of weight of 35 pounds in the course of 38 weeks, but also a considerable improvement of hearing.

A striking instance, which we have met with among our patients, is that of a man who after a hernia operation received the following "strengthening diet" at home:

At 6:00 a. m.—250 g. of chocolate,
At 7:00 a. m.—150-200 g. of coffee,
At 9:00 a. m.—1,000 g. of Jersey milk (plus breakfast),
At 10:00 a. m.—250 g. of broth.
At 12:00 a. m.—350 g. of Bavarian beer (plus lunch),
At 3:00 p. m.—250 g. of broth,
At 4:00 p. m.—350 g. of ale (plus dinner),
At 9:00 p. m.—150-200 g. of coffee or tea,
Altogether about 2,800 g. of liquid plus the meals proper.

After two months of this diet he suffered his first attack of violent giddiness. The man recovered after a dehydrating diet, during which he lost 4.4 kg. in weight. At the same time his hearing, which had been greatly reduced, returned to normal.

Such a development of giddiness, partial deafness and buzzing in the ears may likewise be observed after the forced intake of liquid during lactation, or after drinking-cures for urinary affections or rheumatism.

The injurious influence of cold and damp air, which was mentioned by Hippocrates, and the beneficial influence of dryness and warmth may possibly also have a certain relation to changes in the discharge of liquid.

In this connection it should be mentioned that varying postures and the consequently varying hydrostatic pressure in the vascular system frequently assert themselves very distinctly. Numerous patients feel poorly in the morning, particularly after a long, sound sleep. Their buzzing, giddiness, headache and vasomotor rhinitis are stronger, and particularly their deafness is worse, which can be measured by tests. At the same time the skin of the face, and especially of the eyelids, frequently presents edematous changes, being coarse or wrinkled. In the course of the day these symptoms decrease, the hearing improves considerably, whereas the infiltrations of the legs increase during the erect posture. There are also patients who, when lying down during the day, experience buzzing in the ears, or giddiness.

In the following communications* we shall endeavor to supplement these clinical observations by the results derived from a series of experiments.

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V

THE PERIPHERAL ENDINGS OF THE COCHLEAR NERVE*

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Pathologic changes within the cochlear nerve and nerve endings are important findings which may be associated with functional impairment of the neural mechanism of hearing. The nature of the degenerative changes in the fibers of the cochlear nerve have been described for a large group of agents, including certain drugs. But little attention has been paid to the nerve endings on the hair cells within the organ of Corti. This is because demonstration of the nerve endings requires the use of difficult technical procedures which do not always result in satisfactory preparations. Several investigators have described the appearance of these endings under normal conditions for human and animal material with the use of different silver impregnation methods. De Nó[1] has summarized the results of the previous studies.

Degeneration of the myelin sheath of the cochlear nerve fibers when produced by experimental methods is usually first apparent for the fibers in the osseus lamina spiralis and modiolus of the cochlea.[2] Similar changes appear later in the fibers of the auditory nerve. The effects of various agents such as drugs, bacterial toxins and missing dietary factors should be correlated with histopathological changes in the cochlear nerve and other lesions in the different parts of the peripheral auditory mechanism.

From previous studies it seemed probable that nerve endings on the hair cells might reveal some of the early changes in the cochlea for such a drug as quinine. The present studies are concerned with the appearance of the nerve endings after supravital staining with methylene blue for control and drug-injected animals.

PROCEDURE

Fragments of the dissected cochleæ (membranous) of six guinea pigs weighing 110 to 250 gms. were stained with a 1:5000 solution

*From the Laboratories of the George Williams Hooper Foundation, University of California, San Francisco.

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of methylene blue. These animals were not injected with a drug and the appearance of the nerve endings and hair cells of their cochleæ served as controls. Each of three guinea pigs weighing 250 to 300 gms. received three subcutaneous injections of quinine (300 mgs. per kg., 2½% solution) over a period of ten days. The cochleæ of these animals were dissected and the material stained with methylene blue in the same way as for the control group.

The animals were decapitated and the temporal bones dissected free of muscular attachments. The bulla of each ear was opened and the cochlea removed. Soft tissues in the cochlea were separated from the bone under a dissecting microscope and with the use of fine needles. Further dissection of the material was made on a glass slide in warmed physiological saline and pieces transferred to other slides and immersed in several drops of a 1:5000 solution of methylene blue (National Aniline Co.). After allowing various intervals for staining to occur a cover glass was placed over each slide and the degree of staining observed under the microscope (3mm. objective, 10X ocular). Camera lucida drawings of certain hair cells and their nerve endings were made, using a magnification of 810 diameters.

RESULTS

Many attempts were necessary to determine the sample and dilution of methylene blue which gave consistent and satisfactory staining in each instance. The dissection of the material was found to be an important procedure because of the tendency of the hair cells to break up and lose their identity after too extensive manipulation.

In a study of the controls, four minutes after the application of the dye there was found to be a slight coloration of the protoplasm of the cell with a deeper tinge to the nerve endings (Fig. 1a). Nine minutes of staining resulted in a more intensely stained cell and a fibrillar network within the cytoplasm became visible. The nerve endings were deeply stained (Fig. 1b), as well as the nucleus and certain fine granules in the cytoplasm. Eighteen minutes produced an intensely stained external hair cell (Fig. 1c) with considerably less contrast in the coloration between nerve endings and protoplasm of the cell. Figure 1d is a camera lucida drawing of an internal hair cell which illustrates the staining after eight minutes. These cells do not appear to stain so intensely as the external hair cells in the same length of time. Aside from their slight difference in shape, they do not seem to show the same degree of cytological differentiation as the external hair cells reveal.

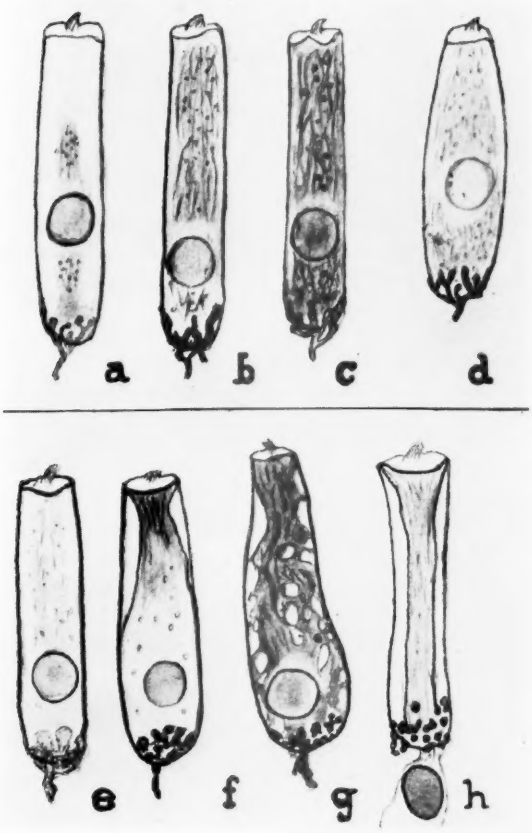

Fig. 1. Camera lucida drawings of external and internal hair cells and nerve endings in the lower half of the cochlea of control and quinine injected animals. Material was supravitally stained with a 1:5000 solution of methylene blue for various intervals of time. Magnification 810x.

a. An external hair cell from a cochlea of one of the controls after four minutes of staining. The nerve endings are in evidence and there is a slight tinge to the protoplasm of the cell.

b. A similar cell from another control animal which was stained for nine minutes. The nerve endings and protoplasm of the cell are more intensely stained than for a.

c. Another external hair cell from the same cochlea as b after eighteen minutes of staining. There is less contrast between nerve endings and cell protoplasm than for shorter periods of staining.

d. An internal hair cell from the same cochlea as b and c, after eight minutes of staining.

e. An external hair cell from the cochlea of an animal which received quinine injections for ten days. This cell was stained for six minutes. The nerve endings are swollen and stain faintly as compared to the controls.

f. An external hair cell from the same preparation as e, in which the nerve endings have lost their continuity and appear to be intensely stained granules after twelve minutes.

g. An external hair cell from the cochlea of another drug injected animal which was stained for ten minutes. More marked degenerative changes are apparent than for cells e and f.

h. An external hair cell from the same preparation as g after ten minutes of staining. The nucleus is extruded from the base of the cell.

Figure 1e is an outline of an external hair cell from the cochlea of one of the animals which received quinine over a period of ten days. It will be noted that the nerve endings appear to be swollen at their terminations and less intensely stained after six minutes than for a cell from a normal animal which was stained for four minutes (Fig. 1a). It is probable that this may represent the earliest discernible change in the degeneration of the nerve endings due to the effects of quinine. In Fig. 1f the nerve endings are broken up so as to give a granular appearance and are more deeply stained than in the preceding cell. Marked degenerative changes for the cytoplasm as well as the nerve endings are illustrated in Fig. 1g. The extruded nucleus illustrated in Fig. 1h may be the result of dissection on a cell which is damaged by the effects of the drug. It is of interest, nevertheless, to associate this loss of nucleus with the observations of Lurie,[3] who regards the loss of the nucleus for the external hair cells in embedded and sectioned cochleæ as indicative of the effects of quinine.

The histopathologic findings described were not apparent in the material studied for the guinea pigs which did not receive the drug. The changes were more pronounced in isolated groups of hair cells from the lower half of the cochleæ of the drug-injected animals. Not all hair cells from the same area appeared to be in an identical state of degeneration. A single cell, such as illustrated in Fig. 1g, was found in the vicinity of other cells of which nerve endings and protoplasm were considerably less damaged. That the nerve endings reflected the degree of change in the hair cells with which they were in contact was obvious.

COMMENTS

Degenerative changes in the hair cells and the nerve endings presumably result in faulty reception of stimuli and their proper transmission. Thus it is possible that one of the earliest alterations in the neural mechanism for the transmission of impulses may be found in the condition of the hair cells and their nerve endings. It is impossible to judge accurately, by the use of ordinary technical methods, the condition of these cells, except in advanced stages of degeneration for which the organ of Corti is lacking or the external hair cells obviously damaged. A study in which the finer cytological changes in the cochlea[1] are correlated with hearing tests in the same animals should determine their functional significance.

Certain aural disturbances such as tinnitus, for which there is at present no recognizable lesion in the peripheral auditory mechan-

ism, may finally be explained by alterations of the nature described or by other similar changes. Unfortunately this symptom is not detectable in animals by present hearing test methods.

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VI

THE INNER EAR FROM AN EXPERIMENTAL AND CLINICAL STANDPOINT*

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The present discussion will be confined entirely to that part of the inner ear concerned with auditory sensation and not with the vestibular apparatus. The extraordinary delicacy of this apparatus and its amazingly designed structure have made it a fascinating field of investigation both for physiologists and otologists since Corti's organ was first described. In the past few years, as new methods of experimentation have been developed, great impetus has been given to scientific effort in this particular field of research. Few problems present more interesting material to the investigator than does this part of the auditory apparatus. In the same way its importance, from the standpoint of the clinician, has been emphasized repeatedly in recent years with an increasing appreciation of the röle remote factors and conditions may have upon the development of impaired hearing. It is no longer possible to deal with inner ear deafness solely by prophylaxis against possible infection or the elimination of infection already present; nor can specific procedures designed to increase the efficiency of an end organ already impaired be the rational therapeutic effort. Established physiologic principles together with carefully analysed clinical data must furnish the foundation upon which any diagnostic structure is to be built.

RECENT EXPERIMENTAL WORK

With the development of the electrical method of investigation and a wider knowledge of the technic of reflex conditioning many investigators have endeavored to demonstrate with scientific accuracy the truth or fallacy of theory and hypothesis which had so long obtained as the only explanations of true cochlear function. The interrelationship of the various parts of the neural mechanism of hearing is so intimate that analysis of individual function presents almost insurmountable technical difficulties. However, several im-

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portant facts have already been demonstrated with satisfactory conclusiveness.

In 1934 and 1935 the majority of American investigators devoted their entire attention to the problem of cochlear localization and the resonance theory[1]. Though a mass of material has been presented the final proof of true localization has yet to be brought forward. Various compromises have been suggested such as the spread effect under increased intensity of stimulation, but the critical experiment, loss of low frequencies by apical damage, has yet to be demonstrated. High frequencies are invariably affected no matter where an experimental lesion may be produced in the cochlear spiral. Frequencies are lost in direct proportion to the number of ganglion cells destroyed beginning always at the highest and spreading downward.

Measurements of the so-called cochlear effect indicate that high frequencies are registered with greater intensity at the base and low frequencies at the apex, while the intermediate response ranges between these two points[2]. These results must be accepted with certain reservations so far as actual hearing is concerned. The cochlear response, however measured, can be completely dissociated from true action potential and in consequence less weight can be given these findings than would otherwise be the case.

Measured changes in intralabyrinthine pressure and the effect upon hearing have been made.[3] Pressures increased far beyond physiologic limits have little effect upon the function of the inner ear, while lowered pressures produce an immediate impairment throughout the entire auditory range.

More recently the effect of various drugs upon the organ of Corti has been studied and again with damage or destruction of the basal structures high frequencies are promptly lost. Indeed any chemical substance, in moderately high concentration, applied to the delicate round window membrane will damage the organ of Corti immediately adjacent with great promptness .

In experimental sections of the auditory nerve, whether complete or partial, all observers have noted the importance of maintaining an intact blood supply[4]. If this precaution is successfully taken the organ of Corti will remain intact regardless of the extent of degeneration of the spiral ganglion. Partial interference results in atrophy of the organ of Corti; complete interference results in necrosis and an obliterative labyrinthitis. Any collateral circulation which may exist is entirely inadequate for any practical purpose.

Changes in acuity due to acute blood pressure changes are being investigated at the present time though there is little reason to feel that such changes, certainly where increased pressures alone are concerned, would have any profound effect. Sustained low pressures might very well result in a definite impairment. Animals with an experimentally produced chronic hypertension must be studied for possible effects, both microscopic and functional, on the auditory apparatus.

Specific autonomic control of structures of the inner ear is highly problematical. Except for the general pressor effects of certain drugs no satisfactory evidence is available to indicate that interference with the cervical sympathetics exerts any influence upon the sense of hearing.

Experimental studies of bone conduction, though somewhat limited in scope by technical difficulties, indicate nevertheless that a profound reduction may occur as a result of many factors other than actual nerve lesions[5]. It has been demonstrated, for instance, that the ossicular chain is an important route for the conveyance of bone conducted stimuli. In general air conduction and bone conduction are changed by experimental procedures in the same direction if not to the same extent and true increase of bone conduction is seldom if ever observed.

CLINICAL OBSERVATIONS

For this brief review of available experimental data it can be seen that clinical deafness might well result from transient change in the inner ear and as such should be susceptible to a properly designed and directed therapeutic effort. Again, as in experimental studies, localization in the strict sense has not been conclusively proven by any clinical observations. The masterly study of Crowe, Guild and Polvogt[6] shows definitely that with increasing destruction of the spiral ganglia and organ of Corti high frequencies are lost in direct proportion to the extent of the pathology. In this series, however, no case of apical atrophy was found and consequently no conclusion as to its possible effect can be reached. Operative section of the vestibular division of the nervus acousticus might well furnish data, but up to the present time no histologic material is available to demonstrate the effect of possible damage to all or any part of the cochlear division of the nerve. It is well known that cochlear atrophy may be associated with advanced age decades. Therefore, being a definite organic change our interest is more academic than practical. The condition is one which must be accepted, therapy is unavailing.

If we accept the thesis that decreased bone conduction is not necessarily the criterion of actual nerve atrophy we must search for other methods of diagnosis to determine beyond question whether the impairment is due to a true organic destruction of the neural mechanism of the ear or whether we are dealing with a condition in which the observed deafness is but a single manifestation of a general constitutional condition.

Recently two new diagnostic methods have been developed which offer considerable promise in the general effort to arrive at accurate diagnosis. It has been shown that deafness due to a conductive lesion protects the ear from fatigue. When the deafness has been caused by partial nerve atrophy the remaining intact elements will fatigue quite readily if exposed to appropriate stimulating intensities.

Loudness balances, as suggested by Fowler[7] and Steinberg[8], give important information to be used in the diagnostic procedure. The method, however, is not infallible, is somewhat limited in scope and requires more equipment than is available to the average otologist. In the majority of cases it is a definite differential aid in the establishment of an accurate diagnosis.

During the past few years recognition of new etiologic factors in hearing loss has developed in an extraordinary way. Allergy, though probably never directly responsible, by its influence upon sinus susceptibility, maintenance of low blood pressure and possibly autonomic manifestations, certainly renders the individual less resistant to adverse conditions. Acute infectious conditions with their damaging neural sequelae, drug poisoning in which individual idiosyncracy plays such an important part are now regarded as important factors in so-called congenital deafness. Nutritional influence must very definitely be considered and the vitamin content of diet be evaluated in individuals who are constitutionally inferior. Occupational hazard has long been recognized as a definite cause of impaired hearing and this factor is responsible for large pension budgets in manufacturing concerns. Yet one important executive was uninterested in the possibility of prevention because he had gone through all the various branches of his business and at seventy-five had only the hearing loss that might be expected at his particular age.

Endocrinopathies, as they become better understood, will undoubtedly be found to have an important bearing upon deafness. This effect, however remote, must surely be, in some one of its aspects, neural in origin. There is little doubt that correction of a clear cut hypothyroidism may improve the general well being of the individual

to an extent sufficient to improve hearing which is definitely impaired. The case of a musician who exhibits all the classic symptoms of hypothyroidism is illustrative. Deafness had begun to interfere seriously with his work as an orchestra leader. Under appropriate thyroid therapy fatigue was no longer a constant sequel of his day's occupation and his audiogram during courses of treatment shows a definite tendency towards improvement.

Dyspituitrism associated with deafness must be considered a toxic phenomenon rather than a true organic condition. The case of a young woman with characteristic skull changes, an almost completely obliterated sella, a long history of susceptibility to chronic infection of mucous membrane, respiratory and visceral, is typical. Appropriate specific therapy and operation complicate a true analysis of improvement, but at any rate experience would suggest that compensation for the glandular lack must have aided at least in the ultimate result of any operative procedure.

The more recent (Ciocco[9]) analyses of central nervous system lues indicates that the auditory nerve is not really susceptible to damage by the spirocheta pallida. This is certainly true of acquired syphilis whatever röle this organism may play in congenital or hereditary deafness. This particular situation is probably unique in medicine, and in consequence a source of some satisfaction, for in almost no other condition involving neural damage can syphilis be relegated to so inconsequential an etiologic position.

The most interesting and illusive group of cases from a diagnostic standpoint are those of completely unknown etiology and yet possessed of a symptom complex which is practically unmistakable. The outstanding example of this group is Ménière's disease, probably a true toxic neuritis of the vestibular division of the auditory nerve. No adequate pathologic studies have as yet been reported. Dandy's large series of operative cases has happily not yielded a single nerve for postmortem examination. A paralyzed sense organ, limb or muscle can certainly not be the site of neural excitation, yet elimination of the toxic factor might well be more desirable than cranial surgery. Medical treatment, though less dramatic and possibly less certain of result, should unquestionably be instituted and given reasonable trial before the last resort to surgery is made. Unfortunately the leading exponents of medical treatment have neglected to follow the symptomatic improvement with careful audiometric studies. The two must of course go hand in hand and improvement in hearing might well be a deciding factor in ultimate prognosis.

Vestibular symptoms, by their very nature, are sensational, while progressive or even sudden deafness is a matter of much less moment. Cases of cochlear nerve involvment, entirely free from vestibular symptoms do occur, and therapy is effective for the particular disability presenting provided the diagnosis is arrived at with adequate precautions.

DISCUSSION

However much one might hope for help in this particular discussion from experimental findings, at the present time little of concrete value from the clinical standpoint is available. The Resonance theory and localization is of minor importance. Auditory fatigue, an inner ear phenomenon, the division of bone conduction findings into conductive and neural elements, intralabyrinthine pressure changes, are the sole contributions of practical interest so far as clinical otology is concerned. Detonation experiments, exposure to high intensities of sound, the effect of drugs have added little to observations already made in the course of careful routine clinical examinations. The entire subject of toxic neuritis of the cochlear division of the auditory nerve is definitely a matter of great uncertainty. Many factors undoubtedly contribute to impairment of function of this particular cranial nerve and to the neural mechanism of its end organ. The known microscopic pathology of deafness is a matter of relatively gross change. The situation of the end organ in its unique bony encasement restricts, a priori, the finer histologic techniques involved in any demonstration of slight cellular and nuclear changes. Clinical diagnosis must be the final resort and having arrived at that, the accuracy of the study must be gauged by the success of the therapeutic measures employed. Organic change or destruction should be as obvious as it is irremediable. When such a change cannot be demonstrated beyond any reasonable doubt the situation challenges every resource of the otologist's diagnostic acumen.

CONCLUSIONS

1. Impaired bone conduction is not necessarily an indication of atrophy of the nerve or organ of Corti.

2. Loudness balances and fatique tests are valuable adjuncts in differentiating between nerve and conductive deafness.

3. Toxic neuritis of the cochlear division of the eighth nerve undoubtedly plays an important role in the development of certain cases of deafness.

4. Accurate determination of the etiologic factor is usually possible and appropriate therapy will arrest the progress of the impairment and in many instances bring about improvement.

HYPOTHYROID

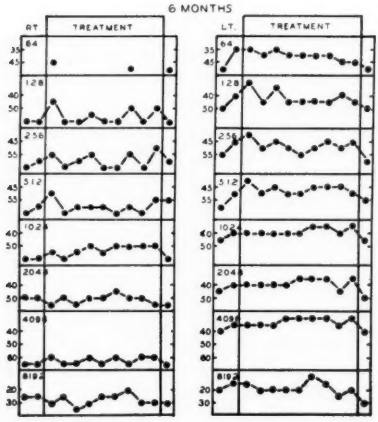

Fig. 1. Composite audiogram of a patient with rather marked hypothyroidism. Following the institution of thyroid extract therapy this patient had marked subjective improvement and, as shown in the audiogram, actual improvement of hearing during the period of treatment. The improvement was greatest in the left ear, which, at the time of the first examination was approximately 10 decibels better than the right for all frequencies. There was a prompt falling off for all frequencies when treatment was discontinued.

MÉNIÈRE'S DISEASE

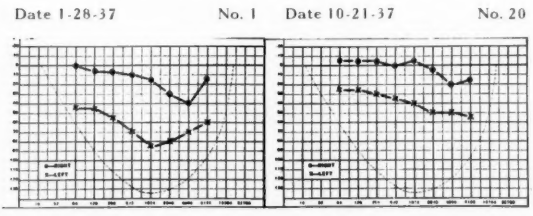

Fig. 2. Audiogram of a patient with typical Ménière's disease. Following dietary treatment the attacks became less frequent and much less severe. He has been entirely symptom free for six months. During this period there was a striking improvement in hearing and complete disappearance of tinnitus. The audiograms were made at the time of patient's first visit and ten months later. Appropriate masking was used in all tests. During a period of hospitalization attacks could be precipitated at will by administration of sodium chloride.

DETONATION

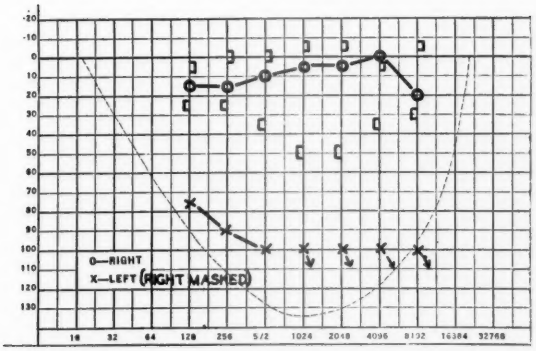

Fig. 3. Audiogram of a patient whose left ear became completely deaf after explosion of a firecracker. AC and BC right are practically normal. Note that the four high frequencies are absent both by air and bone, while the three lower frequencies are still present. The damage done to the cochlea by detonation is not restricted to particular areas. Islands of normal organ of Corti can be found at any point on the spiral and yet the high frequencies are always lost.

TOXIC NEURITIS TOTAL

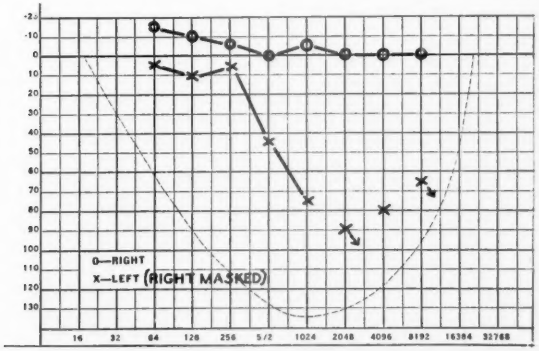

Fig. 4. Acute toxic neuritis of infectious origin characterized by extreme vertigo and loss of hearing on the left side. Note loss of high frequencies and normal low frequency recording. This patient has a dead labyrinth on the left side. It is unlikely that such a toxic condition would be selective so far as the cochlea division of the nerve is concerned, but rather that sufficient nerve fibers remained with intact organ of Corti to transmit the lower frequencies.

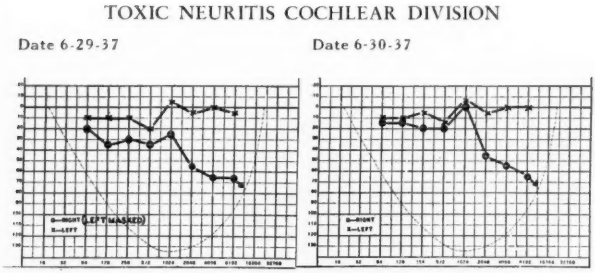

Fig. 5. Acute loss of hearing in the right ear not associated with vestibular disturbance or acute infection. Vestibular tests were normal and an eighth nerve tumor could be ruled out satisfactorily. On the day of the second audiogram a series of laboratory tests were made and the patient had several vomiting attacks. Note striking improvement in the affected ear while the normal ear remained unchanged. A third audiogram made after readjustment with cessation of the vomiting showed that the left ear had returned to its original low level. Low salt diet was the obvious therapy.

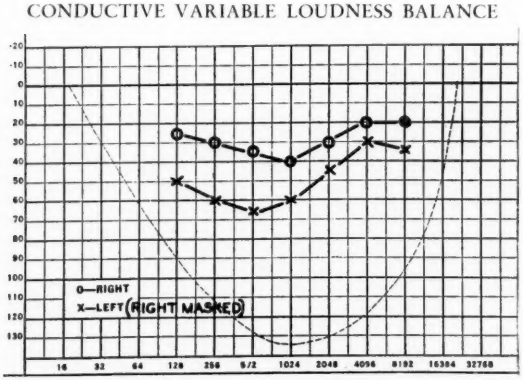

Fig. 6. Conductive deafness with normal bone conduction. Loudness balances made on this patient in the routine diagnostic study were of the typical variable type indicating inner ear involvement. Though this anomalous response is unusual the case emphasizes the importance of a general survey in all studies of deafness. No means of diagnostic study should be neglected in the effort to arrive at an accurate diagnosis.

LOUDNESS BALANCE

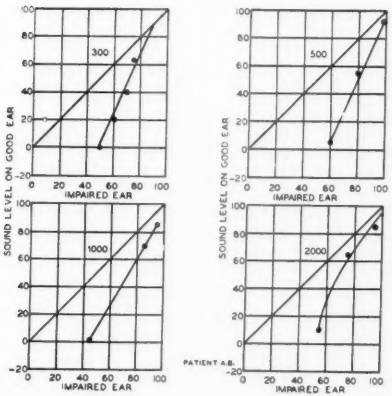

Fig. 7. Typical loudness audiogram of the variable type in a patient with a toxic neuritis. At high intensities of the four frequencies used the patient heard equally well in both the normal and impaired ear. Note that with each increase of intensity the charted line or impaired ear audiogram approaches more closely the diagonal line or level of the normal ear.

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VII

LATERAL SINUS THROMBOSIS

A Study of 88 Cases, with 10 of Venous Thrombosis, Found at 12,500 Consecutive Necropsies*

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PART I

SINUS THROMBOSIS

A detailed study was made of 1,000 cases of cerebral vascular lesions found at 12,500 consecutive necropsies done at Los Angeles General Hospital between April 15, 1918, and November 1, 1934. Of this number 133 (13.3%) were found to harbor cerebral embolic lesions; 418 hemorrhagic processes, and 449 thrombotic pathologic conditions, among which 98 (9.8%) were sinus and venous thromboses.

These 98 cases of cerebral venous system involvement have been reviewed with an exposition of the comparative incidence of the various types of lesions, of the contributory factors and etiological sources in their production and of the sex, age and survival periods in the patients demonstrating these types of intracranial pathology.

Lateral sinus thrombus was found present in 38 (38.8%) patients, the sinus on the left being involved in 25, on the right side in 10 and bilaterally in three. The longitudinal sinus was involved in 16 (16.3%) patients, the cavernous and its accessory sinuses in 34 (34.7%) and venous thromboses were disclosed in 10 (10%).

Other coexisting cerebral vascular involvements are indicated in the accompanying charts and a number of brief history abstracts have been included to demonstrate the varying contributory factors and etiological sources, the symptoms presented and the extent of

*This study is a chapter from a monograph, "Vascular Encephalopathy," submitted as a thesis to the Graduate Medical School of the University of Pennsylvania as a partial requirement for the doctorate degree in Neurology, granted June 9, 1937.

Presented in abstract at the Eye and Ear Section of the Los Angeles County Medical Association, February 28, 1938.

pathologic involvement disclosed at necropsy. A number of illustrative cases with photographs of the cerebral pathology are presented.

The history of the development of knowledge regarding these conditions has been reviewed from the literature with particular attention to diagnostic procedures, the sources and pathways of infection and the opinions regarding the surgical treatment.

Hoopis,[1] in 1826, was the first to point out the relationship between suppurative mastoiditis and sinus thrombosis. Zufal,[2] in 1880, advocated opening the lateral sinus and ligating the internal jugular vein, which he did in 1884. Horsley,[4] independently in 1886, advocated similar treatment, which was popularized three years later by Ballance and Arbuthnot Lane.[5]

Bacteriologically, the streptococcus is the causative agent of sinus thrombosis in 60 per cent of the cases, according to Seydell.[68] Streptococcus mucosus is especially virulent and characterized by late exacerbations with fatal outcome. In chronic cases of anerobes and faculatative anerobes may be the causative organisms. In the majority of sinus thromboses the infection is from one organism. Hayman[59] found in 90 per cent only one organism was causative when the blood culture was made from the blood of the involved sinus. A pure culture was grown in 75 per cent from the smears taken from the thrombus.

Careful technique is necessary for good bacteriological results. The culture is best taken at the height of the temperature or at the termination of a chill if present. Libman[35] got seventeen negative blood cultures in twenty-six cases.

Fient's[10] statistics show the sigmoid sinus was involved in 79 per cent, the bulb in 12½ per cent, the petrosal sinus less frequently and the cavernous least often. The dura veins, the mastoid emissary vein, and in children, the petrosquamous sinus may also become thrombosed. The bulb is usually involved in acute infections and the sigmoid sinus more frequently in chronic inflammation of the mastoid cells. The bulb may also be infected from the labyrinth through the internal auditory vein and the inferior petrosal sinus, as well as by way of the carotid plexus, in which instance no surgical mastoiditis is present. The cavernous sinus may be involved directly by extension of the infection from the tip of the petrous portion of the temporal bone or indirectly because of thrombosis of the veins of the carotid plexus. Sinus involvement may also be caused by a labyrinthitis, especially if paralabyrinthian cells exist, and also from the

saccus lymphaticus. Trauma may also be etiological in the presence or bacteremia. Thrombosis may occur by intravenous extension through a tributary vein.

There are two theories relative to formation of thrombi. Talkes[14] and others hold it is not necessary for the bacteria to penetrate to or through the intima. The chemico-toxic action of the germs present in the wall of the sinus may produce the clot.

Leutert[10] and others hold that inflammatory changes in the intima are necessary, that germs produce toxins which lower the bactericidal properties of the blood, making the formation of thrombus easier, and that all thrombi are infected from the beginning. Hayman has shown that either or both methods may produce thrombi and the thrombus may be sterile at first and later infected. The formation of a thrombus is a protective measure.

Bacteria may penetrate the sinus wall and not produce thrombosis. Obliterating thrombus is usually laid down in layers and organizations and suppurative destruction then takes place at the same time. Hemorrhage from a sinus is rare, but fatal cases have been reported in mastoiditis complicating scarlet fever and tuberculosis. Spontaneous recovery from sinus thrombosis is possible with obliteration of the sinus or recannulation occurring.

Metastasis is reported from the literature by Seydell[68] in 40 per cent of the cases of one group and in another series in 50 per cent. The localization of metastasis following otitic pyemia, he classifies as follows: (1) Cranial with intracranial metastases and into the eye, nose, paranasal sinuses and pharynx and tonsils. Pia and arachnoid and brain abscesses have been reported. (2) Thoracic-pleural, pulmonary and the heart, especially endocarditis. Lung abscess is more frequent in the chronic types, may be single or multiple and is usually located near the periphera and in the lower lobes. In one series reported, 88 per cent had lung abscesses. (3) Abdominal, producing peritonitis and abscess of the bladder. (4) Peripheral, with metastasis in the skin, bursae, muscles and joints. There is no regularity in time, number and virulence and the metastatic involvement. It is possible for the metastases to develop six to eight weeks after the lesions in the mastoid and sinus have healed. Alexander states bacteria may be found in the blood for months after the infection in the sinus is apparently well. Anatomic variations in the cranial venous sinuses is common. In a study of fifty skulls Edwards[64] found 46 (92%) had a difference in the diameter of the outgoing venous sinuses of the two sides. Twenty-five were larger

on the right side and twenty-one on the left. The discrepancy in size may reach 4.1. In cases where the larger sinuses were on the right, the variation in size was greater than when those on the left side were larger.

Law[51] presented an anatomic specimen of the right temporal bone which showed a strongly developed emissary vein running upward and posteriorly in a sulcus 6.5 mm. wide. The sulcus of the sigmoid sinus was entirely absent; the blood from the right transverse sinus emptied into the right emissary vein and not into the jugular bulb. From the emissary the communication was with the superficial cervical veins direct. He quoted Frey and Hoffman as having reported a similar observation in 1924.

An accessory sinus of Kelck in the roof of the eustachian canal was shown at autopsy in a case of mastoiditis on which Stewart[58] had performed a radical mastoidectomy. The patient had died of cavernous sinus thrombosis. In a report on the examination of thirty-one skulls, Kopetzky[41] found this sinus was present in twenty-three. He called attention to the importance of the presence of fetal blood sinuses like the one described, the petrosquamous and the petro-occipital, as pathways for the spread of infection in the endocranium.

Hayman[59] discussed otogenous systemic infection occurring without sinus thrombosis and cautioned no such diagnosis can be made unless autopsy has shown all cranial sinuses to be free from thrombi. He based the opinion on experimental work that bacteria can penetrate the sinus wall and lead to a sepsis without producing a thrombus in the sinus itself.

German[52] reported two cases of sinus thrombosis with afebrile courses. He states there is no sepsis if the thrombus is solid and sterile or if, while centrally infected, it is solid at both ends and undergoing organization. The general factors that contribute to a lack of a septic clinical process consists mainly in the decreased resistance of the patient, noted particularly in diabetes.

Brunner[53] differentiated two types of sinus thrombosis—that present at the time of the primary operation but which remains latent for a time and then makes itself evident, and that which sets in after operation. In 154 cases of sinus thrombosis, 22 occurred postoperatively and 11 terminated fatally.

Brunetti[54] discussed otogenic thrombophlebitis of the superior petrosal sinus. He stated a chronic suppuration in the epitympanic

portion of the middle ear can erode the bone and by direct continuity cause a thrombosis of the superior petrosal sinus which ends in this vicinity. Isolated at first, the infection soon spreads to the sigmoid or cavernous sinuses when it can be associated with a Gradenigo syndrome. Owing to the venous communication between the petrosal sinus and the apex of the petrous pyramid, the prognosis in these cases is bad, due to the inaccessibility of the area.

McKenzie[55] described two types of bulb thrombosis, the primary bulb thrombosis, which is a direct extension from the middle ear, and a secondary bulb thrombosis, which is an extension of a thrombus within the sigmoid sinus. The only possible way to differentiate the two is by opening the sigmoid sinus. When no blood comes from below, after incision into an apparently healthy-looking sinus, the diagnosis of primary bulb thrombosis is certain. In these he recommended first trying drainage by means of a catheter forced through the jugular vein and lavage of the bulb. He also curettes the bulb both from the sinal and jugular end.

It has been shown by McKernon[25] that one of the essential factors in the diagnosis of primary bulb thrombosis is a normal mastoid process. He considered it possible to establish the diagnosis preoperatively by the presence of sepsis, the decreasing red blood cell count and hemoglobin and the normal condition of the mastoid process in association with a purulent middle ear.

Gondelman and Binstak[57] stated that primary bulb thrombosis can be cured only by an early radical Grunert bulb operation, and that the patient has a chance of recovery only when the vein, bulb and sigmoid sinus are laid open as a groove. He states ligation of the jugular vein alone is insufficient.

Kopetzky[41] does not agree with this dictum, as the majority of patients upon whom he operated recovered after incision of the sinus, ligation of the internal jugular vein and suction and curettage of the jugular bulb. He pointed out the danger a continued focus of sepsis will set up by extension into the inferior petrosal sinus or the condylar veins. He believes the establishment of drainage of the jugular bulb is in itself insufficient for cure. If the condition is recognized and operation performed early, before the thrombus has extended into the communication channels, recovery is spectacular. On the other hand, once the septic focus has extended into the inferior or petrosal sinus or condylar veins, it is impossible to irradicate the lesion thoroughly by operation, even when a bulb operation is performed.

Ricard[56] states traumatic rupture of the lateral sinus may occur at the knee of the sinus, chiefly in fracture of the skull, and epidural hemorrhage ensue. The characteristic symptoms are coma, stertorous breathing, progressive hemiplegia, extensive edema of the temporo-parietal area, dilated pupil on the injured side and slow pulse. Trephining over the area soon locates the source of the hemorrhage and if it is stopped the patient may be saved, otherwise he dies of increased intracranial pressure.

Ottenberg[49] in reporting a case, suggests that an aid is offered in the study of sinus thrombosis by determining a difference in the number of bacteria in the blood of the two internal jugular veins, as obtained by puncture through the skin.

With increased intracranial pressure there may be changes in the eye grounds appearing as optic neuritis and choked disc. In 162 cases Blau[19] found eye ground changes in 69 per cent. The disc changes may be unilateral or bilateral, and when unilateral involvement is present, it usually occurs on the involved side, but may be contralateral. When bilateral the eye signs are usually more pronounced on the affected side. Crowe and Beck's maneuver of compressing the jugular vein produces edema of the disc. An important but unreliable sign is pain and swelling in the region of the jugular vein. When the bulb and jugular vein are affected the ninth, tenth and eleventh cranial nerves may become involved, giving hoarseness, dyspnea, slow pulse, difficulty in swallowing and spasms in the distribution of the spinal accessory nerve.

Halipre and Petit[36] discussed this uncommon phenomenon and reported an example in a child. Bilateral otitis media with sinus thromboses had first caused bilateral facial paralysis from compression of the facial nerves. Then followed thrombosis of the left superior and inferior petrosal sinuses, of the right lateral sinus and jugular vein and finally of the ophthalmic veins with development of exophthalmos. Typical glosso-labio-laryngeal paralysis was present. The pathological involvement was demonstrated by a careful post-mortem examination.

In applying the Tobey-Ayer test in twenty-five operated cases, Seydell found proper localization in seventeen, in six the involvement was not found on the side toward which the test pointed, and in two it was found on the opposite side.

Seydell recommends complete exenteration of the mastoid and exploration of any sinus fistulæ. The sinus plate should always be removed in the presence of septic symptoms and the sinus exposed

throughout the sigmoid area. The jugular vein should be ligated before or after removing the thrombus, depending upon the patient's condition and the condition of the thrombus where no free return of blood is obtained from the bulb. Alexander,[50] who advocates primary ligation, claims a 22 per cent mortality in 155 cases of all types, irrespective of thrombosis. In Haymann's series, 27 per cent were lost where thrombosis was found and ligation was not done. In his 300 cases of otitic sepsis of 33 per cent of the acute cases which were ligated, 95 per cent lived. In the chronic group 52 per cent were ligated with 60 per cent recoveries. The mortality in those not ligated was 28 per cent. This does not prove it is better not to ligate, as the cases requiring ligation were more severe, and those which had no ligation were mild, hence were not to be compared. The mortality now is about 20 per cent, according to Alexander, with variations from 40 to 60 per cent. Death is usually due to bacterial action on vital organs, resulting in pneumonia and meningitis frequently, and occasionally brain abscess.

Eagleton[30] reported circulatory disturbances following ligation of the internal jugular vein in sinus thrombosis from the literature and included one of his own. In his patient, profuse bleeding from the mastoid wound, following ligation, resulted in the immediate appearance of an intense optic neuritis. Failure to relieve the sepsis was due to inability to manipulate the lower end of the sinus because of severe bleeding and the locking in of septic material by the firm tamponage necessary for its control. The non-septic thrombosis of the superior longitudinal sinus and the accompanying meningitis were caused by stagnation of the blood in the sinus, while the secondary hemorrhage, escape of cerebrospinal fluid and development of herniation of nearly the entire left cerebellar hemisphere were the result of increased pressure and weakening of the dura through the firm and prolonged tamponage, all being due directly or indirectly to sudden disturbances in cranial circulation brought about by ligation of the jugular vein.

Rohrback,[8] in 91 cases of ligation for various conditions, found six presenting symptoms of circulatory disturbance. Swelling and edema of the soft parts with cyanosis of the face were found; in four severe headaches occurred and three deaths were undoubtedly due to disturbances of cerebral circulation. G. Fischer[33] observed headache and hemiplegia which later subsided. Osmus[33] reported convulsions and opisthotonos lasting twelve minutes. Winthrop Mitchell[29] observed delirium and unconsciousness which continued for several days. Edema and swelling of the neck and face was

reported by Berard.[23] Richards[24] observed transient mania in one and secondary papillitis in another.

Rohrbach reported continued semicoma for six days and death. At postmortem examination the transverse sinus and jugular vein of the unligated side were found to have lumina the size of a needle and a quill, respectively. In the middle of the right frontal lobe a softened area the size of a five-mark piece was present, and one the size of a two-mark piece in the left second frontal convolution with the cerebral surface covered with numerous smaller areas.

Linser[22] observed delayed return of consciousness in a patient, with death the next day. Hyperemia and edema of the brain were shown. The capacity of the ligated jugular vein was ten times that of the unligated jugular and the transverse sinus on the unligated side was very small. Kummer's[13] patient did not regain consciousness following the anesthetic. Edema of the brain had resulted from effusion of the venous hyperemia and small multiple hemorrhages and extravasation of blood into the ventricles was shown. Grunert's[15] patient did not recover from the narcosis and the brain showed a subdural hemorrhage over the left convexity and hemorrhagic infiltrations around the central convolutions. Hölscher[11] had a death following operations the second, fifth and twenty-first days. Death resulted from thrombosis of the superior longitudinal sinus and brain abscess, the patient having recovered from the immediate circulatory disturbances.

Dench[18] had a patient die with serous meningitis, cerebellar softening and hemorrhage into the spinal cord. Pause[7] reported hemorrhage in the cerebrum with softening. A 7 cm. hemorrhage in the occiput extended into the temporal lobe and the corpus callosum and corpus striatum showed hemorrhagic infiltration. In Streit's[12] case, thrombosis of both lateral sinuses and the right jugular bulb had occurred, a softened area appeared in the cerebellum and there was hemorrhage over the left cerebral hemisphere.

In the examination of 1,022 skulls, Linser[22] found 29 (3%) with the jugular foramen so small the vein could not have been over ¾ mm. in diameter. Of these, 2½ per cent were on the left side and ½% on the right. Cunningham[16] states the internal jugular vein is sometimes smaller or larger than normal, requiring compensatory changes on the opposite side or in the external jugular vein of the same side. One lateral sinus may be absent or very small. Knott[3] found the right lateral sinus is generally larger than the left, but in two instances it was only a small venous canal 1.5 mm. in

diameter following its course to disappear in the mastoid foramen. In four of forty-four specimens the superior longitudinal sinus turned directly into the right lateral sinus, which appeared to be a direct continuation of its fellow. In these instances the left sinus was only one-third the size of the right lateral sinus.

In seventeen skulls Eagleton[30] found the jugular foramen almost absent in one and greatly contracted by septa in two others. Zuckerkandl[13] found a right jugular foramen eight times the size of the one on the left, and Biddle[33] described contracted left and right jugular foramina so small a bristle hardly passes, but the mastoid foramen was 1 cm. in diameter.

Circulatory disturbances in the presence of sinus thrombosis are practically unknown in spite of such abnormalities, as the thrombus is a slow growth allowing adjustment to the great amount of blood to be carried by other channels, so that when the occlusion becomes complete there is little or no disturbance.

Particular care should be observed in ligating the jugular vein in the presence of optic neuritis. Sprague[33] refers to a case of total blindness from optic neuritis in the presence of sinus thrombosis, and Richards[24] reports blindness following ligation for sinus thrombosis.

Theoretically, Eagleton points out, there are certain conditions possible in sinus thrombosis which make ligation of the internal jugular vein dangerous by preventing the return circulation: (1) When the thrombus does not entirely occlude or when it does not extend fully down to the bulb—the inferior petrosal sinus and condyloid being still patent. In either the ligation may cut off the main venous return channels. (2) When the external jugular vein is ligated during the operation—a frequent occurrence because of its course, thus destroying one of the now most important channels. (3) When the ligation of the jugular is below the facial vein or the facial itself is ligated. If several of these conditions occur and the opposite jugular vein is small, cerebral disturbances are apt to follow. To prevent a disturbance of return circulation: (1) Before ligating, make as large an opening in the sinus wall as possible, avoiding the possibility of mistaking a parietal for an occluding thrombus, and reduce to a minimum probing of a disseminating thrombus. (2) If the thrombus is so low this is impossible, application of a clamp for temporary compression of the carotid and with no extra bleeding presenting from the diploic and other small veins it is reasonable to assume the circulation has not been dis-

turbed. (3) Avoid injury to the external jugular vein in the ligation. (4) Ligate above the facial vein, and when this is not possible, the indication for a lower site being chosen is presented.

Watson-Williams[70] considers there is no contraindication to ligation of both jugular veins. He had ligated both jugulars at one sitting in one patient and within a week in several. All did well.

The aid offered by x-ray of the lateral sinus and jugular foramen to interpretation of intracranial complications resulting from venous circulatory disturbance of the temporal bone was recently discussed by Ersner and Myers.[71] They conclude that x-ray of the mastoid alone is insufficient.

The examination should consist of complete study of the temporal bone, including the mastoid process, the petron, the vascular structures and the jugular foramen. An extra small jugular foramen is present on one side in 3 per cent of skulls. The lateral sinus on the same side is always smaller or may be completely absent in this situation. The right lateral sinus is larger than the left in 89 per cent of skulls and the left lateral sinus may be the larger. The right lateral sinus is involved by thrombus more frequently than the left in the proportion of three to two. Complete absence of a lateral sinus has been substantiated by x-ray preoperatively. The presence of a large emissary vein may indicate an absent or small lateral sinus or a thrombus obstructing the sinus. Anomalies of the sinuses and jugular foramen are frequent. During health the circulation is adequate and hence no symptoms ensue. When there is a circulatory disturbance, symptoms appear. Changes in the venous pressure directly influence intracranial pressure which may be increased or decreased. Vasostasis, as a result of pathology of the temporal bone, plus inadequate venous structures, will produce increased intracranial pressure and secondary meningeal symptoms. In carefully correlated x-ray, clinical and operative findings, the knowledge gained from x-ray examinations disclosing the size of the venous structures, has helped in the prognosis and treatment of otitic complications. A stormy clinical course may be suspected when there is a marked difference in the size of the lateral sinuses and where the larger size is involved. Where the infection is in the smaller sinus the prognosis is better and resolution is more apt to occur.

Papillitis is present in 10 to 25 per cent of the cases of sinus thrombosis. Should the papillitis be absent it may be assumed circulation is adequate. The presence of adequate venous circulation plus a papillitis and choked disc indicate increased intracranial pres-

sure towards which measures should be immediately instituted to reduce. The Queckenstedt test should be used, but is only of value when there is a rise in cerebrospinal pressure. A positive Toby-Ayer response must be carefully evaluated. It is possible Gradenigo's syndrome may be present, caused in part by venous disturbance.

Abnormalities in the size of lateral sinuses is again pointed out by Coates, Ersner and Persky.[61] The right is usually larger than the left and one may be absent. The left may be so narrowed as to be a serious factor when ligation or other surgical procedures are contemplated. A typical symptomatology may be due to anomalies in the sinus structure, involvement of the bulb as a primary bulb thrombosis, involvement of the petrosal and other sinuses, and lastly, due to uncommon invading organisms such as streptococcus mucosus capsulatus. Blood examinations, including Schilling hemograms and sedimentation tests, are valuable adjuncts to diagnosis and especially prognosis. Ligation of the jugular vein with exenteration of the infected sinus is the generalized mode of therapeusis. To this they add small, frequent blood transfusions and intravenous therapy of such medicaments as Pregl's iodine, neutral acriflavine, metaphen and mercurochrome. Dick's scarlatinal and polyvalent autostreptococcic sera should also be included.

In their series of 969 mastoidectomies done in Graduate and Mt. Sinai Hospitals, Philadelphia, 1.4 per cent developed lateral sinus thrombosis with a mortality of 14.3 per cent.

In a study of fatal complications of otitis media, with particular reference to the intracranial lesions in 10,000 autopsies, Courville and Nielsen[73] described the variety of manifestations and widespread complex pathological findings which may occur in otogenous thrombophlebitis of the intracranial venous channels. In infants, the additional factors favoring primary thrombosis—anemia, malnutrition and dehydration—may result in widespread occlusion of the venous channels draining the cranial vault, with characteristic changes in the substance of the brain. Thrombosis of the lateral sinus was present in 35 of the otogenous thrombi of the intracranial venous channels, being unilateral in 31 and bilateral in four. Extension into other venous channels occurred in eleven of these, septic meningitis was present in twelve and subdural abscess in three. The superior petrosal sinus was involved four times unilaterally and bilaterally twice each, while the inferior sinus was involved in three, bilaterally in one of them. Otogenous involvement of the superior longitudinal sinus was found in seven, usually the result of retrograde extension in the lateral sinus, more frequently on the right side.

The general symptoms of infected sinus thrombosis are headache and vomiting and frequently chills and fever of a remittant type. There is usually some swelling of the optic discs and the superficial scalp veins may be engorged. The jugular vein may be palpably thrombosed and is often painful. Pain in the chest or abdomen may be evidence of extension of the infection into the general circulation. The marantic type produces more vague symptoms. Headache and signs of intracranial pressure are common. Great variability in the depth of stupor and the localizing symptoms are characteristic. Hemorrhages may produce new symptoms at any time, including the signs of subarachnoid bleeding, according to Grinker[74]. The marantic thrombi occur most frequently in children and the aged. Severe protracted diarrhoeas in infancy predispose to venous thrombosis. Marantic thrombi are prone to occur in the longitudinal sinus, but they may extend from this source to the other sinuses and the cortical veins.

LATERAL SINUS THROMBOSIS

Lateral sinus thrombosis was found to be present in 38 patients, 43.7 per cent of all sinus occlusions, and 8.7 per cent of the instances of all thrombotic processes in this survey. The left lateral sinus was occluded in 25, the right in 10, and bilateral involvement occurred in three. Of the entire group, 26 were males and 12 females, all Caucasians, 14 being two months to nine years of age; six, 14 to 19; two, 23 and 28; four, 31 to 37; four, 41 to 46; seven, 50 to 59, and one, 82, averaging 26.1 years. Their hospital residence varied from eight hours to 37 days, averaging 12.3 days. The survival periods from the onset of cerebral symptoms until death were from two to forty-eight days, averaging 16.4 days.

Coma marked the onset of the cerebral involvement in eleven patients, following convulsions in two of them, accompanied by muscular twitching in two others, and by delirium and irrationalism with the abating depth of coma in three of them. Three patients demonstrated hemiparesis, due to thrombosis and thrombotic softening in two of them. Marked ataxia was the first evidence of the cerebral involvement in two in whose cerebelli encephalitis and an abscess had formed, one of the patients being so dizzy he was unable to stand. Vomiting and nausea were the first symptoms in two patients. In the remainder of the group the symptoms could have been attributed to the otitis media and mastoiditis present, except for neck stiffness and irregular pathological plantar reflexes found in four of them.

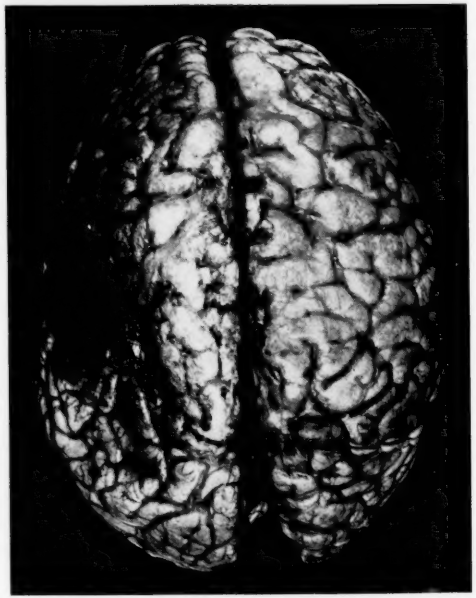

Fig. 1

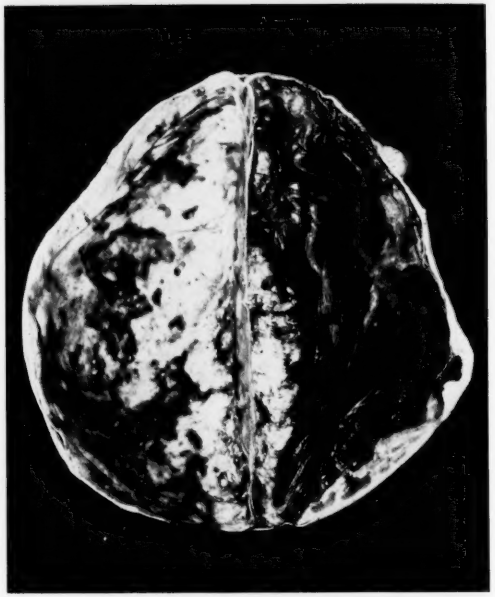

Fig. 2

Figs. 1 and 2. (Lat. S. T. 41). Subarachnoid and subdural hemorrhage following lateral sinus thrombosis.

LEFT LATERAL SINUS THROMBOSIS

The left lateral sinus was involved in 25 (28.1% of those with sinus thromboses) of the 38 patients with lateral sinus thromboses and varying cerebral pathology and meningitis. The right middle cerebral artery was also occluded in a male aged 54 (S. T. 20) who had a gastric carcinoma. The basal ganglia were bilaterally involved by thrombotic softening also in the brain of an 82-year-old female (S. T. 35) whose cerebral basal vessels were extremely sclerotic. Left frontoparietal subdural hemorrhage co-existed in the brain of a male of 50 (S. T. 41) whose heart showed mitral stenosis and endocarditis. The left internal carotid artery was thrombosed in a boy of 15 (S. T. 25) who had bilateral mastoiditis with thrombosis of the left lateral sinus extending to the confluence of the sinuses. The inferior petrosal sinus was also occluded in another patient (S. T. 31), the left cavernous sinus also in one (S. T. 23), and in two (S. T. 33 and 36), localized cerebellar encephalitis and abscess formation and a septic basal meningitis were also present. Meningitis had developed in five others in conjunction with the left lateral sinus thrombosis. Anomalous sinus formation was present in one patient (S. T. 33). In six patients the internal jugular vein contained an extension of the thrombus which extended into the innominate vein in one (S. T. 32) of them. Otitis media was the etiological source of the sinus thrombosis, except in the first three just noted, in 22 of the 25, with mastoiditis having developed in eleven of them. The otitis media had followed a peritonsillar abscess (S. T. 19), measles (S. T. 21), scarlet fever (S. T. 37), and diphtheria (S. T. 38), in one each and was present bilaterally in a 2-year-old diabetic (S. T. 40) in whom multiple pulmonary abscesses and bacillary dysentery were present.

RIGHT LATERAL SINUS THROMBOSIS

The right lateral sinus was thrombosed in ten patients, 11.2 per cent of the cases of sinus thromboses. The cerebellar veins were also thrombosed with resultant hemorrhage in one (S. T. 47) and pial hemorrhage from pial venous thromboses with softening of the underlying brain substance was found in another brain. (S. T. 45). Meningitis was also present in five patients. The internal jugular vein contained extension of the thrombus in four, which had extended into the innominate vein in one of them. Otitis media was the etiological source of the process in all but two patients; one, a male of 50 (S. T. 49), who was admitted to the hospital in coma with lobar pneumonia and died within eight hours, and the other (S. T. 42) who

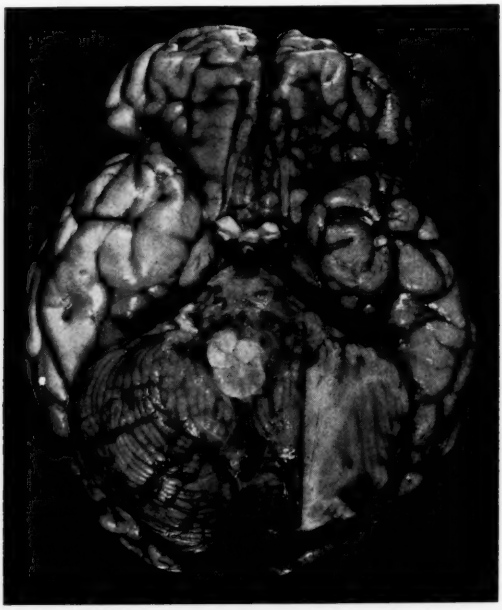

Fig. 3

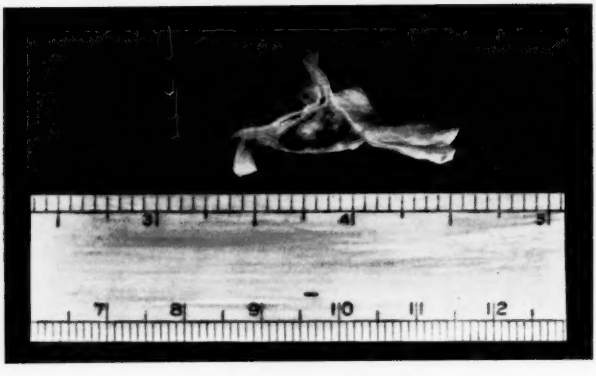

Fig. 4

Figs. 3 and 4. (Case 21). Thrombosis left lateral sinus with pachymeningitis and basal meningitis.

had a nasal furuncle a week when swelling about his eyes appeared five days before his death.

BILATERAL LATERAL SINUS THROMBOSIS

Both lateral sinuses were involved in three patients (3.36%), a 69-day-old female (S. T. 52) with bilateral otitis media and a left temporal abscess, a male of 50 (S. T. 53) with a posterior neck abscess, and a male of 28 (S. T. 54) with left otitis media and mastoiditis, who suddenly became unable to stand on account of dizziness. A 2.5 cm. abscess in the left cerebellar hemisphere explained the dizziness and incoordination present in this last patient.

REPORT OF CASES

CASE 1. (S. T. 52.) The two-months-old babe was admitted in coma with a history of rhinitis and cough for ten days previously. The right temporal region was swollen and fluctuant. Myringotomy and incision of the right temporal abscess resulted in free drainages, but diffuse pulmonary signs developed and death occurred the third day.

A 2 cm. pus pocket was found under the right temporal fascia. From the right sigmoid sinus pus extended down into the right jugular bulb, filled the right lateral and cavernous sinuses and continued posteriorly through the confluence of sinuses around to the left lateral sinus, stopping at the jugular vein origin. The right middle ear contained frank pus and the left a small amount. Smears disclosed the presence of staphylococci. No meningitis was present. The lungs contained mutiple pyogenic abscesses.

CASE 2. (S. T. 53.) A 50-year-old male had noted increasing general weakness and lassitude for four months, had severe night sweats and needed to quit his work as a carpenter. Three months before he was admitted to the hospital an abscess developed in his right shoulder which pointed into the occiput. It was opened a week before he entered the hospital. He was quite emaciated and weakened. The day after admission a chill occurred with a temperature of 103.4° F. following. The liver was enlarged three fingers breadth below the costal margins. The red blood cell count was 4,300,000 and hemoglobin estimation 80 per cent. The leucocyte count was 34,600 with 82 per cent polymorphonuclears. The neck was aspirated with removal of 50 cc. of sanguinous material. The spinal fluid contained 150 leucocytes per ccm. Two days later neck

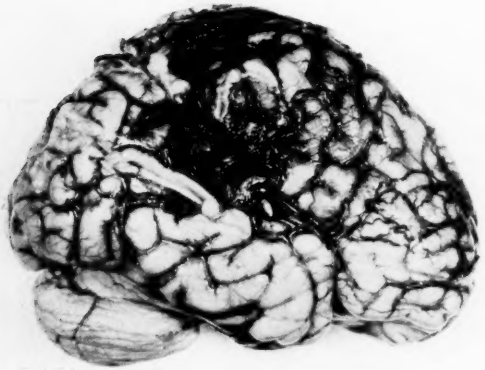

Fig. 5

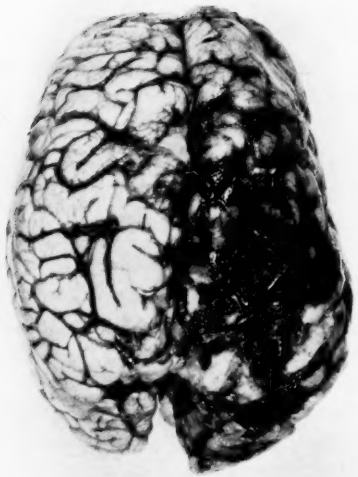

Fig. 6

Figs. 5 and 6. (Case 32). Thrombosis lateral sinus with multiple softening.

rigidity and left Babinski, Chaddock and Oppenheim reactions were obtained. The spinal fluid at this time contained 240 cells, 98 per cent polymorphonuclear leucocytes and a few highly refractile bodies that tended to bud. Smears and cultures were negative. Death occurred the third day after signs of intracranial involvement presented, the seventh day of his hospital residence.

Both the lateral and cavernous sinus on each side were found to contain much sanguinopurulent material with some subdural pus on the right side and four small erosions of the occipital bone from the abscess in the back of the neck. The middle ears were negative and no cerebral abscess formations were present.

CASE 3. (S. T. 54.) A white male of 28 was admitted to the hospital on account of increasing pain in the left ear and mastoid, with such marked dizziness since the previous day that he was unable to stand. Three years previously the ear had discharged for a year. When admitted to the hospital the temperature registered 103.4° F., pulse 116 and blood pressure 110/70. The left membrana tympani was reddened and the left mastoid tender. The neck was rigid and the reflexes on the right side were increased. He had a tendency to fall to the left when in the sitting position. The day after his admission a left mastoidectomy was performed and the left jugular vein was ligated. Death occurred 24 days later.

The right lateral sinus was found to be filled with thrombus and purulent material. There was a 2.5 cm. abscess in the left cerebellum. The left internal jugular vein was ligated at its middle, below which it was empty, but above the ligation thrombus extended through the bulb to fill the left lateral sinus.

ABSTRACTS OF CASES OF LEFT LATERAL SINUS THROMBOSIS

CASE 4. (S. T. 34.) A 9-year-old girl was admitted to the hospital stuporous and irrational with a temperature of 105° F. and a history of spontaneous discharge from the left ear three weeks previously, which had continued since. Three weeks before the rupture of the membrani tympani, measles had developed, and three days later epidemic parotitis. The following ten days pain persisted in the left ear. Examination on her entrance to the hospital disclosed the left ear discharge and bilateral Kernig and Brudzinski reactions. The spinal fluid pressure was 275 mm. of water, cloudy and contained 1,900 cells per ccm. No organisms were found in the spinal fluid smear and no culture growth occurred. She died

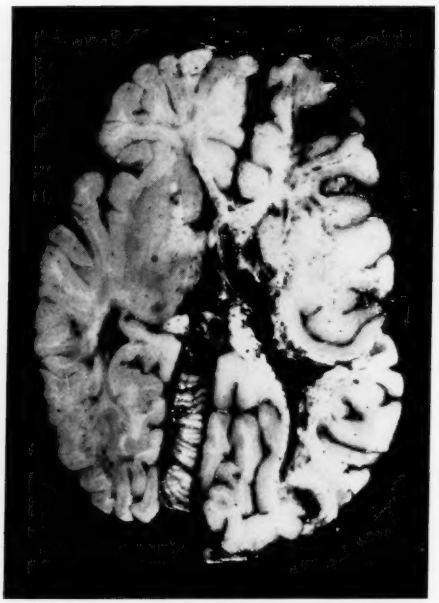

Fig. 7

Fig. 7. (Case 32). Thrombosis lateral sinus with multiple softening.

three days later. At necropsy pus was found in the subarachnoid space at the base in scattered masses. The left lateral sinus contained a blood clot.

CASE 5. (S. T. 31.) Another 9-year-old girl had a history of recurrent otitis media most of her life. Five days before she was admitted to the hospital, pain developed in the left ear with otorrhoea, fever and chills. Vomiting persisted the next three days and the neck became stiff. When brought to the hospital she was rational, the neck was stiff and tenderness was present over the left mastoid and jugular regions, but no Kernig reaction was elicited. The temperature was 101.6° F., the pulse 136, and respiration 28. The urine was negative, red blood cell count 3,300,000, white blood count 21,600, with 88 per cent polymorphonuclear leucocytes and blood culture negative. The spinal fluid pressure was 260 mm. of

water, on right jugular pressure 320, and on left jugular pressure 280. The fluid was clear and contained 260 cells per ccm., 60 per cent polymorphonuclear leucocytes. Sugar and globulin content were normal. At operation a sinus thrombus and cholesteatoma were found. With consolidation in the right lower lobe of the lung the temperature elevated to 107° F. and death resulted the fourth day of her hospital residence. The left lateral sinus was found to contain an antemortem thrombus and the inferior petrosal sinus was filled with dirty-gray purulent material.

CASE 6. (S. T. 28.) Pain in the left ear had been present for a week in a boy of 15 when he was brought to the hospital. He had a history of the ear discharging for 13 years, ceasing a week before with the severe ear pain ensuing with tenderness back of it. At examination ataxia of the left upper extremity was shown and a right Babinski and bilateral Chaddock, Gordon and Oppenheim reactions were elicited. He died three days later when a left intralobar cerebellar abscess was found and the left lateral sinus was filled with clot not a direct extension from the abscess. Green pus was present about the left cerebellar hemisphere with considerable exudate over its superior surface.

CASE 7. (S. T. 32.) Another boy of 15 had been discharged from the hospital three weeks before his readmission, following diabetic coma. A week later left ear pain appeared and a myringotomy was done. Since then his neck had been stiff and sore throat and left-sided headache had been present. Roentgenological report indicated possible left mastoiditis. His temperature was 100.2° F., pulse 90 and respirations 22. The blood pressure was 95/60. Two plus glycosuria was present with 3.5 gm. of sugar eliminated in 24 hours. Acetone was negative. The red blood cell count was 4,200,000, the white cells numbered 9,400 per ccm. with 88 per cent polymorphonuclear leucocytes. The temperature increased to 104.4° F., and at operation following a left lateral sinus thrombosis was found. Signs of meningitis developed the third day afterwards and he died the next day, the thirteenth day of his hospital residence.

At examination of the brain the left lateral sinus about the region of the torcula disclosed a red yellow clot, further down containing pus and continuing as a partially organized thrombus to the jugular bulb. The internal jugular vein was filled with necrosis continuing into the left innominate vein for two-thirds of its course to the superior vena cava, terminating abruptly in the form of a

shell, which apparently had kept the free pus from grossly contaminating the blood stream. Diffuse septic meningitis with abundant subarachnoid pus was present, particularly over the frontal and temporal poles and in the basilar cisterns extending along the blood vessels over the parietal lobes. The left inferior anastomotic and cerebellar veins were thrombosed, but no abscesses had developed anywhere.

CASE 8. (S. T. 19.) A peritonsillar abscess one week before entrance to the hospital had followed acute tonsillitis in a 23-year-old female. The left side of the neck had been swollen and the left ear had been painful for three days. The blood pressure reading was 100/40. The red blood cell count was 5,248,000 cells, the white cell count 29,000, with 89 per cent polymorphonuclear leucocytes when she was admitted, and 17,600 white cells two days later. A slight trace of albumin was reported in the urine. Fever persisted and increased; a blood culture was reported negative, but lobar pneumonia developed and death occurred 19 days after she entered the hospital. At necropsy the left lateral sinus was found to be filled with dry, gray and adherent thrombus from which free pus could be expressed, extending back to the level of the occipital sinus and anteriorly to the sphenoidal sinus. The left jugular vein was filled as far as could be probed. The other sinuses were free and the mastoids clear. The left lower pulmonary lobe was consolidated.

CASE 9. (S. T. 30.) A severe upper respiratory tract infection had been present for five weeks, followed by left ear pain with a sanguinopurulent discharge and left mastoid tenderness for one week in a male aged 31 when he entered the hospital with a temperature of 100.6° F., pulse of 90 and respiration 20. The blood pressure was 112/68. The neurological examination was negative. The blood count showed 5,050,000 red cells and hemoglobin of 90 per cent. The white blood count was 11,200 with 69 per cent polymorphonuclear leucocytes. The blood Wassermann was reported negative. A simple mastoidectomy was done and a left mastoiditis, a left lateral sinus thrombosis and extradural abscess was found. Three days later his temperature was 102° F., pulse 66 and the white blood count 22,100 with 89 per cent polymorphonuclear leucocytes. Spinal fluid examination disclosed 3,850 cells, 88 per cent polymorphonuclears per ccm. The left jugular vein was ligated and two days later the spinal fluid contained 3,400 cells per ccm. and pneumococci were demonstrated in its smear. The temperature increased

to 105 and 106 degrees F. terminally, with death occurring six days after the mastoidectomy was performed and nineteen days after the appearance of the sanguinopurulent ear discharge. The brain examination showed an antemortem thrombus in the left lateral sinus extending almost to the jugular foramen. Just below its sigmoid portion was a small perforation in the dura and an interfoliar foci of pus on the anterior margin of the cerebellum. Basal septic meningitis was present which showed pneumococci in the smears.

CASE 10.—A female aged 33 (S. T. 33) had a left earache and myringotomy a month before left mastoid swelling appeared. Night sweats, high fever and a bloody discharge from the left ear had been present for a week when she entered the hospital. Her blood pressure was 118/72, the urine negative. The white blood count was 14,000 per ccm., 76 per cent polymorphonuclear leucocytes and nine days later 28,700 with 89 per cent polymorphonuclears. A radical mastoidectomy was done with subsequent blood transfusions, but progressive intracranial involvement ensued and she died 26 days after admission to the hospital. At examination of the brain the superior longitudinal sinus was found to empty into the right lateral sinus. The left lateral sinus communicated deeper and was filled with thrombus mixed with pus as far as the torcular Herophi and it was filled with a more whitish thrombus down toward the jugular bulb. The brain was congested and subarachnoid hemorrhage was present about its middle dorsolateral border. There was heavy fibrinopurulent exudate in the basilar cisterns. Subarachnoid hemorrhage appeared on the superior surface of the right cerebellum, the regional vessels being thrombosed and a 1 cm. abscess presented on the median anterior margin of the left cerebellar hemisphere.

CASE 11.—Frontal sinus pain and discharge had been present for four years in a male (S. T. 36) 57 years of age, when six weeks before entering the hospital both ears began to discharge large amounts of pus. He suffered severe aching pains in the frontal and mastoid regions bilaterally and was very dizzy and nauseated. At examination his temperature and pulse were normal and the blood pressure was 150/90. Much discharge continued from the right ear. Blood examination showed 4,001,000 red blood cells and 5,325 white cells, 73 per cent polymorphonuclears, 23 per cent lymphocytes and 4 per cent mononuclears. A simple left mastoidectomy was done the fourth day he was in the hospital and a left jugular ligation ten days later. The spinal fluid was turbid when examined at this time. He died the fifteenth day of his hospital residence.

Brain examination disclosed the presence of a free thrombus in the sigmoid portion of the left lateral sinus as far as its genu. A recent localized encephalitis was present on the lateral portion of the anterior margin of the left cerebellar hemisphere secondary to thromboses in the afferent veins in this region. A septic meningitis, with more abundant exudate at the base, was present. Both middle ears were filled with pus.

CASE 12.—Diffuse thrombotic processes were shown in the brain and numerous organs as well as metastatic carcinoma in a male of 56 (S. T. 20) who fell on the street on account of a sudden left hemiplegia. His blood pressure was 120/80 and the urine contained a trace of albumin. Bilateral purulent otitis media was present and diffuse abdominal pain and digestive distress. He survived the cerebral vascular accident 19 days.

A partially organized thrombus was found occluding the first cm. of the right middle cerebral artery with softening shown in the right lenticulate nucleus and insula with involvement of the anterior limb of the internal capsule near its genu. There was thrombosis of the left lateral sinus and purulent bilateral otitis media, mastoiditis and sphenoid sinusitis present. Gastric carcinoma was shown with metastases in the mesentery, lymph nodes and splenic capsule. Recent thromboses had occurred in the inferior vena cava, the left common iliac and left renal veins, in branches of the left portal vein and in branches to the left kidney and right lobe of the liver.

CASE 13. (S. T. 35.) A female of 82 had been well until a sudden paralysis of the right side and aphasia appeared the evening before she was admitted to the hospital, semistuporous. The left arm and leg were moved but she was unable to speak and right facial weakness was apparent. Conjugate deviation of the eyes to the left persisted. The blood pressure was 180/110 and the temperature 100.2° F., increasing to 104°, with pulmonary involvement. She survived two weeks.

Brain examination showed extreme sclerosis of the basal vessels and an antemortem thrombus entirely filling the left lateral sinus. The mastoids and middle ears were normal. Coronal cerebral sections demonstrated a 2 cm. softened area immediately superior to the upper pole of the caudate nucleus on the left side, a thrombotic lesion in the path of the corona radiata from the left motor cortex and very minute petechial areas of softening in both basal ganglia.

CASE 14. (S. T. 26.) A 14-year-old female had a series of chills two days before she was brought to the hospital, and had been delirious for 24 hours. The head was retracted, the body opisthotonic and the neck rigid. The left tympanic membrane was red and bulging. The spinal fluid was under slightly increased pressure and contained 72 cells per cubic centimeter, of which 60 were leucocytes. The next day the temperature rose to 105 F., and a left mastoidectomy was done. Death occurred the next day.

The left lateral sinus was found to contain a two-inch organized clot. The other sinuses were negative. Smears from the sinus thrombosis showed the presence of pneumococci.

CASE 15. (S. T. 27.) Chills and fever had been experienced by a female of 58 for four weeks before she entered the hospital. There had been left ear and facial pain for six months. When admitted to the hospital the temperature was 98° F., pulse 120, and blood pressure 160/80. A Babinski reaction was obtained on the right side. The white blood cell count was 6,000 with 90 per cent polymorphonuclear leucocytes. The blood sugar content was 185 mgm. per 100 ccm. Roentgenological examination showed the mastoids to be dense. The swelling in the left side of the neck increased in spite of general improvement until her death a month after her admission to the hospital.

A small amount of purulent exudate was present over the cerebral hemispheres and a large amount in the lateral ventricles. The left sigmoid and lateral sinuses contained a thrombus, a direct extension from the left mastoid which extended into the jugular vein. Otitis media and mastoiditis were present bilaterally. There was marked cellulitis with a large abscess formation in the left side of the neck extending anteriorly to the midline at the upper level of the sternum. The left kidney contained an abscess and was surrounded by a perinephritis abscess. Smears from the neck abscess contained Friedlander's bacilli.

CASE 16. (S. T. 37.) A white boy aged 4 was admitted to the communicable disease department on account of scarlet fever with coryza, with the rash persisting for ten days afterwards. Two weeks after his admission myringotomy was performed and repeated in the presence of a developing left mastoiditis. Two weeks later a left mastoidectomy was performed and death followed two days later. Cultures from the mastoid at the time of the operation gave a growth of hemolytic streptococcus.

In addition to the exposure of the left posterior mastoid cells, a 15 mm. square of bone had been removed from over the knee of the left lateral sinus. At this point the sinus contained a small patch of thrombotic material adherent to its intima, but with no further extension in other sinuses. There was pus in the epitympanic and zygomatic cells on the left side and in all of the cells on the right side. Both middle ears contained much purulent material. The brain was negative.

CASE 17. (S. T. 38.) Another boy of five was admitted to the communicable disease department with scarlet fever of 12 days duration. He had been mentally dull from birth and presented a tremor of the head and spastic lower extremities. When examined on admission the temperature was 102° F., pulse 150 and respirations 40. There was a left external rectus palsy present, the neck was rigid and a right Babinski reaction was elicited. A left myringotomy resulted in a profuse purulent discharge two days later, but the temperature had increased to 105° F. Positive diphtheritic culture was obtained from throat smears and 20,000 units of antidipththeritic serum was administered. A rash appeared over the trunk on the same day. The next day the boy was semicomatose and presented opisthotonus. A right myringotomy resulted in purulent discharge. Two days later the lower extremities were tender and swollen and he died the next day, the sixth day after his admission to the hospital, eighteen days following the onset of scarlet fever, and four days after a positive diphtheritic throat culture was obtained.

Flecks of fibrin and pus appeared on the interior surface of the cerebellum and the subarachnoid fluid was slightly cloudy and greenish-gray, friable thrombus, firmly adherent to its wall extended into the jugular vein where it was also firmly adherent. The left middle ear and mastoid contained a large amount of light brown seropurulent exudate. The right ear contained a small amount of pale brown, slightly turbid exudate, but the right mastoid was clear. The sphenoid and right frontal sinuses were partly filled with moderately thick light brown purulent exudate; the right frontal was not involved.

CASE 18. (S. T. 39.) Pain behind the left ear had been increasing for a month when a white female of 59 was admitted to the hospital. A complete left facial paralysis and left mastoid tenderness were present. The temperature was 98° F., the pulse 84, respirations 20, and the blood pressure 160/90. The urine and blood Wassermann reactions were negative. The day after her admission

a simple left mastoidectomy was performed. Sixteen days later she vomited blood and passed tarry stools. Paralysis of the left seventh, ninth and tenth cranial nerves appeared. Nine days afterwards a purpura covered the entire body. The erythrocyte count was 3,730,000, hemoglobin estimation 86 per cent, leucocyte count 16,700 and platelets numbered 82,000. Death occurred on the twenty-ninth postoperative day, two days after the appearance of the purpura.

The dura in the left posterior and middle fossæ was covered with hemorrhagic friable material 1 to 3 mm. thick. The left lateral sinus was filled with blood clot and gray-white to grayish-yellow tumor tissue which also entirely filled and distended the internal jugular vein and foramen. There were many small hemorrhages over the left temporal, parietal and occipital lobes and a few over the left frontal lobe. The cerebral veins over the lateral surface of the left temporal lobe were thrombosed. Carcinoma of the stomach had metastasized to the liver, kidneys, suprarenals, pancreas, the abdominal and bronchial lymph nodes, the vertebrae and base of the skull. Moderate general arteriosclerosis was general.

CASE 19. (S. T. 40.) A two and a half year old boy had been treated in the out-patient department for a year for diabetes. The formula of C 90, P 40, F 70 had been prescribed with five units of insulin A. M. and six units P. M., with occasional glycosuria and acetonuria appearing. Three days before he was admitted to the hospital he became feverish and vomited. Eight extra units of insulin were added to the dosage. The next two days the fever had continued with diarrhoea once an hour. When brought to the hospital the temperature was 99.2° F. The physical examination was negative. The urine contained 2 per cent sugar and 2-plus acetone. The blood sugar content was 168 mgm. per 100 cc. and the carbon dioxide combining power was 25.4 volume per cent. Frequent vomiting continued and the temperature increased to 104.6° F. and the pulse rate to 130. Bowel movements were from four to ten times daily. Repeated stool cultures were negative. The babe was continually on the verge of acidosis or shock in spite of frequent administrations of glucose and insulin in the attempts at their prevention, with several blood transfusions. The fourteenth day after hospital admission a series of convulsions occurred and he soon died.

The left lateral sinus was found completely filled by an adherent laminated, dark red and yellow-gray thrombus. The brain was negative. Both middle ears contained a large amount of yellow

seropurulent exudate. Bilateral multiple lung abscesses were present, and the dysentery was found to be of the bacillary (Flexner) type.

CASE 20. (S. T. 41.) Following pain of increasing severity in the back of his head and neck for a month, a white male of 50 was found unconscious in his room the evening before he was brought to the hospital. A history was obtained of temperature habits and the occurrence of a comatose spell two years before, when he had been found in a downtown building with no remembrance regarding the attack when he regained consciousness the next morning, without residuals. Three months before his hospital admission he had fallen in the bathroom for no known reason and injured his shoulder. The physical examination was negative when he entered the hospital except for the coma and generally increased reflexes. The blood pressure registered 140/80. The urine was normal and the nonprotein nitrogen blood content was 31 mgm. per 100 cc. of blood. The spinal fluid was normal. Ten days after admission he was able to talk and answer questions. A few days later temperature elevation and pulmonary signs indicated development of confluent pneumonia and he died the eighteenth day of his hospital residence.

An extensive subdural hemorrhage was found to cover the left frontal and parietal lobes. A thrombus in the left lateral sinus extended from below the jugular bulb to the torcular herophili. The other sinuses were clear and the middle ears were normal. A mitral stenosis and rheumatic endocarditis were present.

THROMBOSIS LEFT LATERAL SINUS WITH PACHYMENINGITIS EXTERNA AND BASILAR MENINGITIS FOLLOWING OTITIS MEDIA

CASE 21.—A 14-year-old girl entered the hospital complaining of stiffness of the neck and fever. Three and one-half weeks before entry she had tonsillitis. Earache appeared and a few days later the left ear spontaneously began to discharge pus. The neck soon became stiff. A left mastoidectomy was performed and the bone was removed down to the lateral sinus, which was found to be thrombosed. The left internal jugular vein was ligated and the sinus above was packed. Stiffness of the neck persisted and developed bilateral strabismus and choked disc. On admission to this hospital she was irritable but conscious. The temperature registered 103.6° F. Internal strabismus and bilateral weakness of the external recti were present. The right arm was flaccidly weakened. Positive Kernig

reactions were elicited. The white blood cells numbered 20,800 with 90 per cent polymorphonuclears. The spinal fluid pressure was over 300 mm. of water and the cell count 5,400 per cc. The second hospital day, two left occipital and temporal trephines were done to probe for a suspected brain abscess, but none was found. Death occurred five days later.

The skull presented the surgical removal of a portion of the left mastoid. There was a trephine opening in the left posterior temporal area and one in the left occipital region. The middle ear contained fibropurulent exudate. The right middle ear and mastoid were normal. The meninges were congested and on removal of the brain a purulent exudate was seen, with a great increase of fluid over the pons, particularly on its anterior surface. The left lateral sinus was thrombosed and a fresh chicken fat type of clot extended backward to its junction with the longitudinal sinus. The brain weighed 1,475 grams.

Following its fixation, exudate was found in the posterior fossa over the external surface of the dura around the angle of the sinus. In the left lateral sinus extending from below this exudate, was found an organized thrombus which, on cut section, showed a mottled whitish-brown appearance. The convolutions of the brain were somewhat generally flattened and a faint exudate appeared in the subarachnoid space over the dorso-lateral surface. The veins were markedly distended, particularly the superior cerebral and cerebellar veins. These were most conspicuous over the frontal lobes, more over the left. The entire venous system was more prominent on the left than the right. The base of the brain was covered with a heavy fibrinopurulent greenish exudate which obscured the pons and the structures about the chiasm and filled the cisterna magna. In the posterior portion of the second left middle temporal convolution was a puncture mark, evidently surgical, and a similar one appeared in the surface of the cerebellum about 2 cm. from its posterior border. A horizontal section through the brain showed marked dilatation of the lateral ventricles with exudate covering their choroid plexus.

ABSTRACTS OF CASES OF RIGHT LATERAL SINUS THROMBOSIS

CASE 22. (S. T. 42.) A male of 44 demonstrated marked swelling about both eyeballs when admitted to the hospital and was irrational the next day. Three days later left arm and leg weakness was shown. The turbid spinal fluid was under increased pressure.

The neck was stiff, the reflexes were equal and normal, but a bilateral Kernig reaction was elicited. The blood was reported with a hemoglobin of 80 (Sahli), a red blood count of 4,300,000, and a white count of 14,400 with 80 per cent polymorphonuclear leucocytes. The intracranial pathology found included (1) bilateral staphylococcic otitis media; (2) a small abscess formation in the right temporal lobe; (3) septic thrombi in the ophthalmic veins and in the cavernous, superior and inferior petrosal, and both lateral sinuses; (4) subdural abscess diffused over the sphenoid and about the gasserian ganglion; (5) bilateral retrobulbar orbital abscesses; (6) acute purulent basilar staphylococcic meningitis, and (7) purulent sphenoid sinusitis.

CASE 23. (S. T. 48.) Three boys, aged 4, 5 and 8, suffered right lateral sinus thrombosis. The youngest boy (S. T. 48) had been in general ill health and a chronic cough had persisted since a right thigh scald one and one-half years before. Five days before admission to the hospital swelling appeared back of the right mastoid. The next day the ear began to discharge and chills, fever and headache developed. At examination on his hospital entrance the temperature was 104 F. and pulse 120. The spinal fluid pressure was under 12 mm. of mercury and it contained six cells per ccm. Sugar content was normal, globulin was not increased and no culture growth was obtained. The red blood cell count was 3,170,000, hemoglobin 65 per cent, white blood count 17,950, with 90 per cent polymorphonuclear leucocytes. The fifth day of his hospital residence a right mastoidectomy was done and the right lateral sinus was evacuated. Bilateral arthritis of both hip joints had developed and two days after the mastoid operation a large amount of pus was evacuated at an arthrotomy of the left hip joint. The next day the child removed the head dressing with a resulting sudden, large loss of blood and ensuing death. A small amount of antemortem clot was found adhered to the inner wall of the right lateral sinus and some clotted blood was present on the inferior surface of the right occiput.

CASE 24. (S. T. 47.) The right ear had discharged for a week, two weeks before a 5-year-old boy was brought to the hospital unconscious demonstrating coarse muscular twitching. The fixed right pupil was smaller than the left. The right membrani tympani was bulging. Deep reflexes were all increased and a left Babinski reaction was elicited. The spinal fluid was under only 60 mm. of water pressure, but was cloudy and contained 7,000 cells, 86 per cent poly-

morphonuclear leucocytes and 14 per cent lymphocytes. A trace of globulin was present and no sugar reaction occurred with 25 drops of fluid in Benedict's solution. A thrombus was removed from the right lateral sinus at the mastoidectomy done and the right jugular vein was ligated, but death occurred the third day of his hospital residence.

Brain examination disclosed a recent basal meningitis. Several cerebellar veins were thrombosed along the lateral aspect of the left cerebellar hemisphere and considerable softening with punctate hemorrhages were present subadjacently. A recent 3 cm. blood clot was found between the dura and the cerebellar hemisphere. No sinus thrombosis was present, that of the right lateral sinus having been completely removed at operation.

CASE 25. (S. T. 51.) A similar progression occurred in a boy of eight admitted to the hospital with right mastoid pain and swelling after ten days pain and discharge in the right ear. The red, bulging, right membrani tympani was incised and the right jugular vein was ligated two days later. Cellulitis of the left arm developed within a week, evidences of bronchopneumonia were present the next day, and a week later, two days before his death, both legs were swollen and edematous. The urine contained 2-plus albumin. The clinical temperature increased to 105° F. terminally.

At necropsy the right lateral sinus was found to be filled with a firm reddish white clot, and the right sphenoidal sinus was filled with yellow purulent material. Below the site of the right internal jugular vein ligation was a yellow friable clot extending into the innominate vein as far as the superior vena cava. A pulmonary infarct with early abscess formation and empyema was present in the right lower pulmonary lobe.

CASE 26. (S. T. 50.) A male of 19 was admitted to the hospital with right peripheral facial palsy and a history of intermittent right ear discharge for two years, with the last attack two months previously. A radical right mastoidectomy had been done three weeks before he entered the hospital, but he had made poor progress, headache, stupor and delirium developing. Slight right optic papilledema was present and vertical nystagmus presented on upward gaze. The right arm was atonic and ataxic. The neck was rigid and bilateral Babinski and Kernig reactions were elicited. The right cerebellum was explored for abscess, but none found. He died on the fourth post-operative day when the right lateral sinus and jugular bulb were found to contain purulent blood clot. The other sinuses

were clear and no abscess formation was present, the trephine and needling wounds also being clean. The petrous ridge was eroded. The large anastomotic veins on the left were distended and a heavy tenacious septic exudate was present at the base, more on the left side.

CASE 27. (S. T. 43.) A male of 41 was admitted on account of severe right mastoid pain. A right myringotomy had been done five weeks previously with profuse discharge of pus since. The neck was rigid and a right Kernig and bilateral Babinski reactions were elicited. He was mentally clear on admission, but presented various widespread muscular twitchings and rapidly increasing coma ensued. The white blood count was 18,000 with 90 per cent polymorphonuclear leucocytes. The urine contained 2-plus albumen and numerous hyaline and granular casts. Smears from the spinal fluid showed gram positive streptococci. A right mastoidectomy and jugular ligation were done, but death occurred three days later.

The right lateral sinus was found distended and packed but had perforated, giving acute cortical and basilar meningitis. The internal jugular vein was ligated and the sinus clear from the operative procedure. The other sinuses were not involved.

CASE 28. (S. T. 49.) A male, aged 50, was admitted to the hospital without a history and died within 12 hours. His temperature was 102 F., pulse 110, respiration 36, and blood pressure 130 80. Lobar pneumonia and edema of the extremities were present. Bilateral Babinski, Kernig and Brudzinski reactions were elicited. The white blood count was 44,000 with 94 per cent polymorphonuclears. The urine contained a trace of albumin. The spinal fluid was cloudy, yellowish, and under 400 mm. of water pressure. Globulin content was much increased and sugar reduction did not occur with 20 drops of fluid in Benedict's solution. There were 4,400 cells present per ccm. and many gram positive diplococci. A diffuse leptomeningitis with exudate in the cisterna were shown and recent thrombosis of the right lateral sinus. Lobar pneumonia was present and smears from the lung and the meninges showed pneumococci.

CASE 29. (S. T. 44.) Sore throat and left ear discharge had been present one week in a 7-year-old girl when she was brought to the communicable disease department with the rash of scarlet fever. A left mastoidectomy showed the presence of streptococcus viridans. Two days later both middle ears were discharging and a positive

diphtheritic throat culture was obtained. Little change occurred in the patient until ten weeks later, when two severe convulsions occurred and death followed.

A pyothrombosis filled the right lateral and sigmoid sinuses down the right jugular vein to 1.5 cm. above the clavicle. Purulent bilateral otitis media was present but no meningitis.

CASE 30. (S. T. 45.) A four-months-old female babe was brought to the hospital on account of toxic gastroenteritis and diarrhea. Left myringotomy resulted in profuse discharge. The white blood cell count was 26,800 per ccm. Two weeks after admission pneumonic signs developed and death occurred three days later.

The right lateral sinus contained a thrombus with definite grayish organization appearing to be three or four days old. There was some pial hemorrhage about the lateral sinus and over the right temporal lobe. The major right pulmonary vessels were thrombosed.

CASE 31. (S. T. 46.) A history of right ear discharge for three weeks was given in a boy of nine when he was brought to the hospital acutely ill and stuporous. The temperature registered 105° F. The spinal fluid contained 65 cells, 12 leucocytes per ccm. The white blood cell count was 15,800 per ccm. A right mastoidectomy was done. Six days later the spinal fluid cell count was 80 lymphocytes per ccm. and four days after this 540 cells per ccm. A blood culture at this time gave a growth of pneumococcus. Death occurred the fifteenth day of hospital residence.

Fibropurulent material in the right lateral sinus extended in the beginning of the jugular bulb. Left otitis media and mastoiditis were present.

(1) THROMBOSIS OF THE RIGHT LATERAL SINUS WITH RETROGRADE EXTENSION INTO THE SUPERIOR LONGITUDINAL, THE STRAIGHT GYRI, AND THE VENOUS SYSTEM OF GALEN AND INTO THE CEREBRAL VEINS.

(2) EXTENSIVE SOFTENING OF THE BRAIN IN THE RIGHT PARIETO-OCCIPITAL REGION, WITH AN ASSOCIATED SPOT OF SOFTENING IN THE RIGHT FRONTAL LOBE.

CASE 32.—This well developed, well nourished infant, eight months old, was brought to the hospital with a history of diarrhea and vomiting for nine days, fever four days, cough for two days,

and drowsy spells beginning the day before. There had been bilateral otitis media four months before entry. The child was obese, acutely ill, dehydrated and stuporous. Bilateral otitis media was present; the discharge from the left ear was purulent and that from the right sanguinopurulent. The stool culture and urine were negative. The diarrhea continued and the stupor was maintained. The temperature elevated from 101° to 104° F., and to 107° terminally the seventh hospital day, when he died in respiratory failure.

There was thrombosis of the upper part of the dependent portion of the genu and the horizontal portion of the right lateral sinus. This thrombus extended into the superior longitudinal sinus, where it continued at least through half its channel. Extension had also taken place into the straight sinus, into the inferior longitudinal sinus, which in this instance was fairly well developed and also extended into the veins of Galen. The thrombus in most of its extent was of recent origin, but that exposed in the genu and horizontal portion of the sinus showed some necrosis and probably suppuration. The superior cerebral veins emptying into the sinus were also thrombosed and on the brain small flecks of thrombus were seen.

The right cerebral hemisphere was remarkably softened and friable, and over the surface of the brain appeared a subarachnoid hemorrhage. The large veins running over the surface of this hemisphere showed local thrombotic spots and were filled with hard, recent blood clot. There was an isolated patch of subpial and subarachnoid hemorrhage over the right frontal lobe, towards its tip, and the veins here showed patches of fibrinous clot. The area of greatest softening was found in the upper parietal region where the tissue showed the greatest amount of reddish softening following the venous occlusion. There was some thrombosis of the superficial cerebral veins on the left as well as the right side.

Horizontal sections disclosed a number of petechial hemorrhages through the centrum on both sides, but more on the left. Gross blood clot appeared in the substance of the left centrum. There was extensive softening of the posterior part of the right parietal lobe and also an area of red softening in the frontal lobe beneath the cortical area described. The major portion of the cortex of the posterior part of the brain showed softening which extended into the ventricle and petechial hemorrhages were found in the corpus callosum.

The venous system of Galen was filled with dark, recent blood clot. The choroid plexus on the right was filled with blood clot and the choroidal vein was occluded.

LEFT LATERAL SINUS THROMBOSIS (25)

Serial No.	Autopsy No.	P. F. No.	Initials	Color	Sex	Age	Involvement Other Than Lateral Thrombosis
17.	82	120-169	M. O.	White	M.	46	
18.	188	130-275	M. P.	White	M.	34	Left jugular vein
19.	1675	199-421	P. M.	White	F.	23	Left jugular vein
20.	4064	266-364	H. R.	White	M.	56	Right middle cerebral artery*
21.	4133	268-766	P. A.	White	M.	1	
22.	4295	271-979	W. P.	White	F.	6	
23.	4374	274-876	P. L.	White	M.	37	Left cavernous
24.	4583	280-923	A. G.	White	M.	15	Inferior petrosal Internal jugular
25.	5961	73-957	A. G.	White	M.	15	Left internal carotid*
26.	6253	90-678	M. M.	White	F.	14	
27.	8685	210-313	V. T.	White	F.	58	Left sigmoid and jugular
28.	9228	245-775	P. D.	White	M.	15	Left cerebellar abscess
29.	9346	251-567	G. S.	White	M.	45	Left anastomotic vein
30.	9558	257-470	M. A.	White	M.	31	Meningitis
31.	9641	262-763	A. R.	White	F.	9	Inferior petrosal
32.	9767	137-053	M. E.	White	M.	15	Interior jugular, innominate, left inferior anastomotic
33.	9844	267-807	T. B.	White	F.	33	Cerebellar veins, subarachnoid hem.; cerebellar abscess, meningitis
34.	10017	280-560	J. R.	White	F.	9	Meningitis
35.	10124	284-660	K. R.	White	F.	82	Left mid. cerebral perf. br.
36.	10361	296-311	J. McC.	White	M.	57	Cerebellar veins and cerebellar encephalitis
37.	11611	351-092	R. H.	White	M.	4	
38.	11624	358-511	B. C.	White	M.	5	
39.	11698	355-371	S. S.	White	F.	59	Jugular
40.	11705	285-777	R. B.	White	M.	2½	
41.	12299	384-757	D. B.	White	M.	49	Left subdural hemorrhage

RIGHT LATERAL SINUS THROMBOSIS (10)

Serial No.	Autopsy No.	P. F. No.	Initials	Color	Sex	Age	Involvement Other Than Lateral Sinus Thrombosis
42.	855	163-419	C. W.	White	M.	44	Cavernous, petrosal, ophthalmic vein, rt. temporal abscess; meningitis
43.	1351	189-313	D. McM.	White	M.	41	Meningitis
44.	4516	274-479	M. A.	White	F.	7	Jugular, pial, hemorrhage from pial, venous thrombosis
45.	5308	35-794	A. W.	White	F.	4 mo.	Jugular
46.	5390	40-107	B. S.	White	M.	9	Cerebellar vein thrombus and hemorrhage
47.	8361	198-480	D. D.	White	M.	5	
48.	9614	261-827	R. S.	White	M.	4	
49.	9639	263-301	R. D.	White	M.	50	Meningitis
50.	10014	273-873	E. M.	White	M.	19	Meningitis
51.	10528	241-664	A. P.	White	M.	8	Int. jugular and innominate

BILATERAL LATERAL SINUS THROMBOSIS (3)

No. Serial	No. Autopsy	P. F. No.	Initials	Color	Sex	Age	Lateral Thrombosis Involvement Other Than
52.	4601	281-612	C. A.	White	F.	69	Right sigmoid
53.	5322	37-698	A. L.	White	M.	50	Bilateral cavernous
54.	11067	108-583	A. E.	White	M.	28	Left int. jugular Left cerebellar abscess

SEX, AGE AND SURVIVAL INCIDENCE IN THE VARIOUS TYPES OF CEREBRAL VENOUS THROMBOSES

	No.	Per Cent	M.	F.	Average Age	Average Survival (Days)
Lateral Sinus Thrombosis	38	38.8	26	12	26.1	14.4
Longitudinal Sinus Thrombosis	16	16.3	10	6	20.6	13.0
Cavernous and other Thrombosis	34	34.7	22	12	28.4	7.75
Venous Thrombosis	10	10.2	5	5	29.9	4.0
	98	100.	63	35	26.25	10.3

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VIII

THE MANAGEMENT AND TREATMENT OF OTOGENIC MENINGITIS*

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Many otologic purulent lesions terminate in a symptom complex that is clinically diagnosed as meningitis, particularly so, if they run a course not interrupted by spontaneous resolution and cure, or when they remain unchecked by surgical means.

In the past there were many such. As we won hard-fought battles for recovery, the general clinical picture of meningitis was broken down into component parts, and penultimate clinical entities became established and comprehended. To the observant clinician these entities indicated a grave situation which unless adequately handled generally terminated in what was known as meningitis. To handle the various clinical entities that emerged from the background of the symptom complex of meningitis there came into practice diverse surgical techniques. Using these, the few clinical evidences of meningeal involvement which the case presented subsided, and recoveries were recorded and published.

When I first started in otology thirty-two years ago, what a literature existed of otitic sepsis, hardly to be differentiated from the septic meningeal infections which generally terminated them. Then, the recognition of septic sinus thrombosis and its amelioration evolved, and the sum total of fatalities lessened. There followed recognition of labyrinthine involvements and treatment, with a like reduction in fatalities, and the better diagnosis and proper surgical handling of both acute and chronic middle ear infections, including those which ruptured extradurally, added further quotas to the credit side of otology. More recently, the significance which the underlying bone structure plays in predetermining the type and character of the purulent lesions which inevitably will follow bacterial invasion has again aided, in permitting an avoidance of intracranial infection, through modifying the indications for operative intervention, by shaping our conception

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of surgical needs to the exact pathology which we know is developing in a given case. The competent among us are less prone to generalize and standardize our indications, but instead to strongly individualize and particularize our surgical indications. By this means, too, we have been enabled radically to cut short the impending meningeal involvement.

Still more recently, otology has won for itself a knowledge of purulencies in the osseous perilabyrinthine spaces, and these lesions with enclosed empyemas of the so-called apex of the petrous pyramid are so much better comprehended, and a long series of surgical techniques available to handle them. It may be recalled that these penultimate clinical entities are now sharply differentiated from the general meningitis groups of intracranial lesions which follow them, and to a very definite degree, this knowledge has lessened the frequency of occurrence of the ultimately fatally ensuing bacterial meningitis.

These penultimate lesions must all be understood as carrying with them, in the clinical aspects they present, data which not so long ago would have caused them to be recorded and published as meningeal infections. Hence, a very rapid review of the findings in the cerebrospinal fluid in non-meningitic temporal bone lesions would seem a proper introduction to the discussion of the findings in bacterial meningitis itself. All this must be comprehended if we are to be in a position to institute adequate remedial therapy.

In examining a large number of cerebrospinal fluids in uncomplicated otitic cases of simple mastoiditis with no epidural extensions, epidural extensions, apicitic, labyrinthitis, otitic sepsis without demonstrable thrombophlebitis, septic sinus thrombosis and brain abscess, there is a fairly marked difference between cases of primary otitis, that is, one not preceded by an antecedent primary disease, and the general run of secondary otitis which follows the usual upper respiratory, or other general infectious diseases, because in the general infectious diseases there were found changes in the cerebrospinal fluid. In uncomplicated otitis and simple mastoiditis, the cerebrospinal fluid usually simply gives an abnormal number of cells in the great majority of cases, running anywhere up to thirty. The chemistry is normal, and the fluid is abacterial. The pressure is moderate, and very often is not abnormal at all. In simple mastoiditis there may be found, in addition, weak protein reactions. With epidural suppurations that have not been present for a very long time, or are not of large extent, there is an increase in the number of cells; the chemistry is normal and abacterial. In apicitis, the only change noted as a rule,

is a somewhat higher number of cells in the fluid over that of uncomplicated mastoiditis, namely, five to seven per cent of times the cells are increased to over thirty, but otherwise the fluid does not show any additional changes. With a large epidural suppuration, one would expect, and usually find, the fluid normal chemically and abacterial, except that the pressure is increased. The protein reactions in about one-fourth the cases are abnormal. The cytology of the fluid remains normal unless there is a meningeal tissue reaction. The same findings as in mastoiditis are true of apicitis, provided the purulency has not ruptured intracranially. When the latter occurs, but has not caused tissue reaction in the meninges, one gets the same findings that are noted in epidural abscesses from other parts of the mastoid. When meningeal tissue reaction occurs, one gets the picture of meningitis and not of apicitis. Of course, there is an increase in pressure; the cell count varies between nine and two hundred, but the total protein reaction remains weak. The chemistry may show a slight tendency toward meningeal reactions, but remains bacteria free. In labyrinthitis, the pressure does not increase, yet one finds an increase in the cell count, nor are abnormal protein reactions noticed. Chemically it remains normal. In sepsis without demonstrable sinus thrombosis, the spinal fluid is the same as is found in the serous meningitis of which more will be said hereafter, namely, that there is an increase in pressure, but there are no evidences of tissue changes. The chemistry is normal, and there is no bacteria present. In sinus thrombosis, the findings vary greatly with the individual case. In about one-half the cases, there is an intracranial pressure due to circulatory changes, and an increase in lymphocytes is very often present—a picture of protective meningeal reaction. If thrombophlebitis occurs in the very early stages of otitis, the changes in the fluid are greater than when it occurs later in the course of the otitic infection. In the early cases, the cell content of the fluid is increased, and the higher the number of cells, the more predominantly the polymorphonuclear forms occur. There are apt to be chemical abnormalities present. In seventeen per cent of the cases the fluid contains bacteria, but this is usually a finding in cases that will terminate fatally. In brain abscesses, the fluid contents on the whole, give the picture of a meningitis with a high cell count, an abnormal chemistry and may contain bacteria. When the abscess is well walled off and its manifest stage is present with pressure signs direct or indirect, the fluid may be found clear and almost normal in chemistry and abacterial, but the pressure is much increased. If the abscess leaks or ruptures, the fluid contains many cells, is turbid and may contain many bacteria. Then the chemistry too will show abnormal findings.

It suffices, for the purpose at hand, not to describe the cerebrospinal fluid findings in detail in this sketch preliminary to the discussion of the question which most concerns us now—meningitis—but it should suffice to indicate differences in the fluid in its trend away from normalcy, so that it can be differentiated from the fluid findings in a case of meningitis which I shall have occasion to mention hereafter.

It is necessary to remember this data moreover, when I come to detail the specific recommendations as to therapy for successfully handling otogenic meningitis, because each of the antecedent purulent lesions must painstakingly be recognized, and after both diagnosis and differential diagnosis from all other lesions, only then can therapy successfully be instituted to combat it. Only by eliminating each and every type of intervening purulency will it be possible to isolate the lesion we are considering, and recognize it as being located in the meninges, and then proceed to handle it at its seat in the meningeal tissues

Before further developing my thesis for controlling infection in the meninges, it becomes necessary to digress, and recall certain fundamentals regarding the meninges and the cerebrospinal fluid it contains. I will not dwell on the anatomy of the meninges, except to recall that the dural blood vessels are not really grouped as part of the central nervous system blood supply, but belong to the system of skeletal blood vessels supplying the skeletal bones, including the skull and terminating in the dura.

Infections usually follow the blood vessel route rather than the anatomical preformed channels. The latter have been too strongly stressed in our textbook literature, and are, to a great extent, a carry-over from the day when the purulent break through into a definite anatomical structure went clinically unrecognized, only to be found at autopsy, and only then comprehended as the anatomical pathway which the infection traveled. The manner of performing the very autopsy produced factors which handicapped the observers in determining the route traveled by the infection. The removal of the diseased temporal bone with its attached dura in one piece has remedied this fault, and only in a specimen thus made can the blood vessel route to the dura be studied properly, and the role that this route plays in the pathogenesis be comprehended. A specimen from a fatally terminating meningeal case thus obtained, makes it possible to trace the lesion through these vessels and determine the röle they play in the meningitic picture.

There has been a general knowledge of the pressure, physical characteristics, the function, the cytology and the carbohydrate content of the cerebrospinal fluid for some time. in 1912[1] I contributed to this Society an extensive study on the cerebrospinal fluid in an endeavor to find an early sign for practical bedside use in diagnosis. Since then, the chemistry of the fluid, particularly as to its copper reducing content, has become established practice, but while I still maintain that the loss of the copper reducing element in the fluid precedes by a few days the demonstration of bacterial flora, now I know that this sign is not found early enough to be of very great practical help in instituting steps to cure the infection. The exact chemical estimation of carbohydrate present in a successive series of spinal taps gives indication of the development of a bacterial infection. Continuing my chemical studies,[2] advantage was taken of comparisons of similar elements in the patient's blood and in his cerebrospinal fluid, both specimens taken at about the same time, and with infinite care to examine the fluid immediately after withdrawal before changes in its chemistry took place because of intervening time. Even in the time lost going from the bedside to the laboratory, the fluid should be guarded to prevent changes, and the receptacles (test tubes) should be hermetically sealed at the bedside with a layer of paraffin at low melting point as soon as the fluid is withdrawn, so as to conserve it and prevent changes which would vitiate the findings from its examination.

Added to the carbohydrate content estimation we also make estimations of the cerebrospinal chlorides, the carbon dioxide content and the lactic acid content, as well as the P_h of the fluid. These elements were compared with similar ones found in the blood plasma drawn at the same time. In meningitis, the spinal fluid shows marked increase in the lactic acid. The blood plasma in meningitis also shows an increase in the lactic acid due to elevation of the temperature and other factors of general import, but the increase noted in the spinal fluid is more marked than the increase found in the blood plasma; in other words, the general increase of lactic acid in the spinal fluid does not occur proportionately, the rise being most marked in the spinal fluid. To understand the chemical changes which take place in the spinal fluid during the progress of a meningitis, it is necessary that the accepted principle of ionic exchange through physiologic membranes and the devices which are known to aid in maintaining the neutrality of the body fluids be understood. Then only can the significance of the deviations from the normal found during the progress of pathological lesions be evaluated and previously recorded empiric results of many investigators become synthesized

into an integral rational whole. The following factors, therefore, should be noted:

1. There are more chlorides in normal spinal fluid than there are in the blood plasma in the same person. In meningitis this gradient tends to be eliminated and the spinal fluid and blood plasma are more nearly alike.

2. In meningitis there is a decrease in the bicarbonate content of the spinal fluid, the value often being as low as thirty-three per cent below that of the blood plasma.

3. The P_h of the spinal fluid in meningitis is much lower than either that of the normal fluid or that of the normal blood, and it is also lower than the blood plasma taken from the meningitic case.

The normal spinal fluid is practically protein free, the protein nitrogen ranging from 35 to 100 mg. per hundred cubic centimeters, while the protein content of the plasma ranges between 6,500 and 8,500 mg. per hundred cubic centimeters. We may then think of the dividing line between plasma and spinal fluid as one which is permeable to all ions except that of protein. Definite laws govern the ionic exchange which were summarized by Donnan. I need not go into the formulas concerned in this.

Suffice for practical application to comprehend that the ratio of lactic acid in the fluid to that of blood is normally about 1 to 1, and in meningitis the lactic acid rises so that the relation of blood plasma to fluid would be as 1 to 3 or more. Summarizing the ionic exchanges we can say that there is a drop in the chlorides, a drop in the carbon dioxide and a drop in the P_h.

Knowing the normal for each element, and faced with a penultimate lesion which is, however, presenting clinical evidences of meningitis, it is unnecessary to do more than adequately handle the penultimate lesion. If this penultimate lesion fails to evince clinical indications localizing itself, the focal lesion will persist in feeding bacteria into the meningeal tissues, and the dramatic clinical signs of meningitis will overshadow the meager signs coming from the actual causal focus located somewhere in the temporal bone. It is then that these chemical reactions should be studied, because if they then show reactions approximating the normal, it tells the observer that the clinical picture notwithstanding, no actual tissue reactions in the meninges are taking place, but that the toxicity and the symptomatology are to be accounted for by a purulent lesion somewhere in the temporal bone with a clinical picture of meningitis, and a

normal chemical reaction in the cerebrospinal fluid as compared to the blood. It would be a mistake to treat the meningeal symptomatology, and overlook the offending focal factor located in the temporal bone. To overlook this bone focus will cause an eventual fatal termination. On the other hand, to be stimulated by approximately normal reactions in the chemistry of the cerebrospinal fluid as compared with that of the blood, to search for a focal lesion at a time when the ultimate pathology in the meninges is as yet unestablished, leads to invariably finding it, eradicating it, and curing the case. In other words, I am trying to convey a differentiation between a recognition of a meningeal invasion by bacteria from a bacterial infection in the meninges, with meningeal tissue reactions. From a comprehension of these biochemical reactions, such a differentiation is often feasible and practicable.

Another fundamental I desire to recall is the influence of small whole blood transfusions on the chemistry of the cerebrospinal fluid. During the stage of invasion, when the patient may even present bacteria in the fluid, I have found that the administration of small, direct transfusions of whole blood, tend to cause a return of the chemical findings toward normalcy. Recently I added another observation. In a case of toxic labyrinthitis wherein there was so marked a disturbance of the static labyrinth that the patient incessantly vomited steadily for almost a week, gradually losing ground, having intense vertigo, spontaneous nystagmus and an ascending facial palsy; it dawned on me that the intimate relationship between the labyrinthine fluid and the cerebrospinal fluid was such that the chemical changes in the one must logically be followed by similar changes in the other, provided that the natural channels opening from the intradural spaces to the labyrinthine fluids spaces were patent. Acting on this belief, the patient was given a small direct blood transfusion rather than a surgical operation to destroy the static labyrinth, and the symptoms abated. There was an immediate favorable response. The vomiting which had been unyielding to all other medical efforts immediately stopped. The vertigo disappeared, as did also the spontaneous nystagmus, and for the first time in ten days the patient took nourishment by mouth and retained it. After two days the symptoms reappeared, but to a lesser degree, and with less intensity. The facial palsy, meanwhile, markedly improved. A second small transfusion abated them immediately. Meanwhile food was taken and retained and strength gradually returned. A third transfusion was given a few days later, putting the patient on the road to recovery of this particular incident in her case history which was concerned with an entirely different otitic lesion.

Transfusions have long been empirically employed. Often, because in our extremity and without knowing what else to do, they were administered as a last resort to give sustaining factors to the patient. My studies have given me the satisfaction of knowing that the small transfusion given repeatedly at intervals of one to two days brings definite chemical reactions in its train, and when intelligently employed is a great aid in the handling of not only bacterial invasion of the cerebrospinal fluid, but in addition, adds beneficial weight in healing a meningeal infection not too long or too extensively established in the meningeal tissues. Additionally, in the cases where I have employed it, I have noted less "blocking" of the flow, less formation of obstructing factors at the natural ventricular openings. In the last series of cases under observation, the flow was not impeded during the course of the disease.

Lately chemotherapy has added its effect in giving us another therapeutic factor. From the employment of sulfanilamide excellent results are now obtained. Here too, like the use of transfusion, the results are the more brilliant, the truer and surer is the complete removal of the antecedent bone focus accomplished.

Of all the types of pneumococcic meningitis, Type III is the most severe. This is probably due both to the greater pathogenicity of this organism and also to the fact that Type III pneumococcal meningitis is most often of otitic origin; otogenous meningitis in general offering a particularly poor prognosis. Furthermore, specific antiserum has not proven as effectual in Type III pneumococcal infections as it has against other types, such as Type I.

In all there have been only five cases reported of recovery in Type III pneumococcal meningitis. Allman[3] recently reported a recovery and found four other cases in reviewing the literature.

In the case reported by Steinholz and Gleich[4] the meningitis occurred in the course of a post-traumatic subacute purulent otitic media. The meningitis was mild, the spinal fluid cell count reaching only two hundred per mm. The organism in this case was of greatly attenuated virulence, since mice infected with specimens of spinal fluid from the patient did not die.

In these cases then, recovery seems to be ascribed to the low virulence[5] of the organism, and to an increased resistance in the host, established in the course of the existence of a long preceding otitis.

I now add another case to the record, one of pneumococcus Type III which I shall only briefly sketch, because it will be reported in

detail elsewhere,[5] where recovery took place. There were, almost coincidental with the onset of the mastoidal symptoms, signs of meningeal involvement. The therapy consisted of:

1. Removal of a residual bone focus in the zygomatic root of the temporal bone.

2. Repeated small transfusions.

3. Administration by mouth of sulfanilamide daily.

In the details of the case, note that I recovered a very definite amount of sulfanilamide from both the blood and from the cerebrospinal fluid. In giving the sulfanilamide, metahemoglobin estimations were continuously made to control its action and prevent a pathological blood picture from developing. I am including pneumococcus Type III in this report, because I consider the pneumococcus of the streptococcus family.

In commenting upon this case the sequence of therapy, namely, surgical removal of the focus, and then the remedial therapy both hemotherapy and chemotherapy deserves notice.

In a second case (to be reported in full elsewhere), a boy ten years of age presented meningeal involvement coincidental to his mastoid infection. The mastoiditis was handled surgically in the usual manner, and although there were clinical signs, particularly generalized frontal headache, there were no real meningeal signs, and because of the afebrile postoperative period the patient was permitted to leave the hospital for ambulatory treatment. Two days later he was readmitted with symptoms of very severe headache, the pain intensity most marked over the right eye, and double vision. On the day of admission the temperature reached to 105.6° F. At the original admission, roentgen films were taken (as is routine with me) of the petrous pyramid as well as the mastoid, and the petrosal pyramid was then negative. Upon readmission, x-rays of the petrosal pyramid denoted a suppurative petrosal lesion on the same side as the mastoid, which because of the symptoms it presented, was localized as in the ante-cochlear area, that is, in the carotid-apical region. In addition to the temperature and headache, the patient presented nuchal rigidity, but no pathological reflexes. The meningeal signs abated after opening the tympanic cavity and locating a large fistula near the tubotympanic orifice which led deeply into the petrosal pyramid. This, of course, was drained. A spinal tap at time of admission gave 3,000 cells and 95 per cent polynuclears, but no growth of organisms. A second tap two days prior to the operation for drainage of the petrosal pyramid by the Almour technique, the fluid contained a few

cells, yet showed the presence of streptococcus hemolyticus on culture. A culture taken from the exposed dura over the petrosa likewise showed streptococcus hemolyticus on culture. During this stay in the hospital the patient was given sulfanilamide and repeated small transfusions. Although there were irritation signs from the meninges at various times in the long history, the patient was almost afebrile during most of this hospital stay. The meningeal symptoms increased in intensity, and our examinations gave us reason to believe that the focus in the petrosal pyramid was not being eradicated by the drainage. So a radical apicectomy (Lempert technique) was undertaken, and the entire contents of the pars petrosa except the labyrinthine and fallopian canal carrying the facial nerve was removed. Then the clinical signs of meningitis gradually abated, only to appear a little later when the drain in this area was removed. This necessitated again curetting out the granulations from the opened petrosal pyramid and putting in a stiff rubber tube to maintain patency and effect drainage; this tube was left in situ for a considerable length of time. Then the clinical signs of meningitis gradually abated, and under the administration of sulfanilamide and repeated small transfusions and repeated spinal taps the patient recovered completely and was discharged from the hospital, cured. He has remained perfectly well and normal since.

In commenting upon this case thus briefly sketched, the point I desire to make is to draw attention to the fact that although the clinical symptoms were held in check in this case they never actually abated for very long, and they were always a potential danger which became a manifest one as long as there was present in the temporal bone a purulent focus. This, in spite of the transfusions and the administration of sulfanilamide. In other words, our therapy would seem to be ineffective if the original focus is not removed. Even the drainage of the petrosal focus failed to clear the meningeal situation, which only yielded to a radical apicectomy. It is well for us who are using any new drug to bear this in mind, because one cannot expect from a drug more than it can do. It can clear the blood stream, and the cerebrospinal fluid, provided more continuing infection does not feed these body fluids with infecting organisms. Sulfanilamide will clear body fluids infected by streptococci, but it seems not to kill streptococci located in an active bone lesion.

In my desire to limit my surgery to the greatest possible extent and still accomplish a cure, I did the least radical procedure first, and so in succession each procedure was done. It being shown that it failed to accomplish what was desired, the surgery was extended, and successively I performed a simple mastoidectomy, radical mastoid-

ectomy and petrosal drainage, decapsulation of the entire inner table of the mastoid process and tympanic cavity, and then a radical apicectomy before I eradicated the bone lesion sufficiently to permit an efficient reaction from the administration of my hemotherapy and chemotherapy. Any surgery less extensive would have resulted in a fatality. The seriousness of the patient's condition may be surmised by the fact that he entered the hospital on February 25, 1937, and his final discharge was on May 29, 1937.

Finally, another series of observations need record and report. Employing the methods of therapy here outlined, namely, surgical removal of any and all demonstrable purulent lesions in the bone, and the administration of small blood transfusions, either alone or in combination with the administration of sulfanilamide, I began to note a lengthening of the course of illness which the infections under my observation ran. Before the days of sulfanilamide, I had an infant under my care which eventually succumbed, in whom, after surgery was performed and repeated transfusions given, combined with continuous forced drainage of the cerebrospinal fluid by the Kubie method, the case ran a course extending over a few months. During this time the purulency of the fluid abated and the fluid became clear and almost sterile.* At a loss to comprehend why recovery did not follow, after death occurred, autopsy was done and revealed the ventricles lined with a pyogenic membrane within which there was clear fluid, but between which and the brain tissue there was a septic infection so completely shut off from the circulating cerebrospinal fluid that the clear taps were explained and the occasional bacteria found could be considered as a spill-over from the other side of the membrane. The case probably died because of a cerebritis brought on by the brain cells being choked by their own products of metabolism which obviously were not being removed by the cerebrospinal fluid circulating on the other side of the pyogenic membrane. This observation leads me to detail two additional case histories only as sketches of their medical history, because they are too long to report in detail here. In both of these cases there developed a postmeningeal otogenic hydrocephalus.

In one, a female aged 17 was admitted with the complaint of diplopia, headache over both parietal and occipital regions, pain in the lower abdominal quadrant and overdue menstrual period. She

*Toward the end of the case, the fluid was crystal clear, and it was only by the most painstaking search that a few organisms were still to be found in it. It seemed as if these were more like adventitious contaminations than active infecting organisms.

gave a history of a simple mastoidectomy performed elsewhere one month previously, following which the discharge from the ear continued and since which time the severe generalized headache had become progressively worse. Examination on admission showed slight nuchal rigidity, a suspicious positive bilateral Kernig and bilateral and equal hyper-reflexia of the extremities. X-rays showed evidence of a recurrence in the mastoid. X-rays of the petrous pyramid showed a generalized diminution in aeration with no evidence of trabeculation in the apical carotid portion of the pyramid. A revision of the mastoid was done at once, and an exploration for an extradural abscess was negative. The patient reacted well immediately postoperatively, but five days later the symptoms continued together with nausea and vomiting. X-ray of the petrous pyramid revealed no change. On the ninth day all clinical signs disappeared and the patient was discharged from the hospital. Patient remained well for sixteen days, when the excruciating headache reappeared, and three days after this she experienced diplopia and again had pain in her right lower abdominal area. The patient was readmitted to the hospital, and neurological examination was negative except for a hyper-reflexia on the left side. X-rays of the mastoid and petrous pyramids at this time were again negative. The postauricular swelling which was now present was incised on the day of admission and a subcutaneous infection of the wound drained. On the following day examination of the fundi showed definite papilledema with small superficial hemorrhages over the disc and retina. A diagnosis of otitic hydrocephalus or pseudo-abscess was made at this time. Spinal taps performed daily showed an increased pressure but low cell count. Following the third tap when 25 cc. of fluid had been withdrawn, the symptoms abated and the patient began to menstruate (apparently due to the decreased pressure on the pituitary gland). The symptoms alternately abated and reappeared for several days. Following an intravenous injection of 50 cc. of 50 per cent sucrose the headache was relieved. After a four-day interval of well-being the temperature rose to 103, neck rigidity appeared, and the neurological signs of meningitis became marked. A spinal tap done at this time showed an increase in pressure, together with a cell count of 8,950. The patient was then started on a regime of small direct transfusions and sulfanilamide. The clinical condition seemed to improve, the cell count decreased, but the pressure remained elevated and there was an increase in lymphocytes. The patient remained comparatively well for a week, when vomiting and headache reappeared, and a spinal tap again showed a high cell count. Another transfusion was given and the symptoms receded and the patient for the first time had fused vision. The patient has

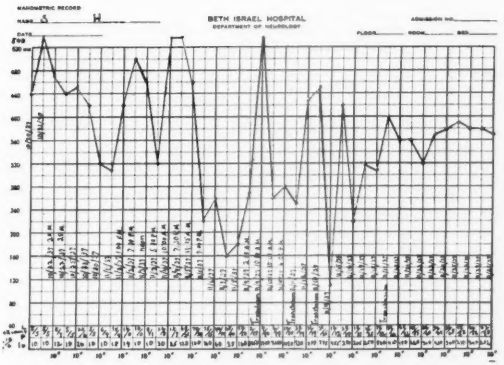

Chart showing degree of intracranial pressure, during the post-meningitic hydrocephalic phases (six weeks period).

continued under the chemotherapy and hemotherapy above outlined, shows no subjective symptoms of increased intracranial pressure, and is now out of bed. Complete recovery has taken place.

In commenting on this case, one may doubt that there is evidence of a bacterial meningitis here, yet in the absence of a pneumatized petrous apex, and knowing that the bone structure of the petrosa was sclerotic, how otherwise to account for the dural changes which produced in their train, the diplopia? How can I account for the meningeal tissue reactions which so distended the ventricles that it gave us an intracranial pressure as shown on the appended cerebrospinal pressure charts?

Furthermore, how account for the increased cerebrospinal fluid cell count? I was sure that the bone as a focus was eliminated as one factor because of the progressive uninterrupted healing of the bone wound, and the return to normalcy of the tympanic cavity and membrana tympani, even while the hydrocephalus continued. I believe that the promptness with which the original mastoid surgery was revised at the very onset of the occurrence of meningeal symptoms, that the removal of the entire inner table of the mastoid process covering the cerebellar and cerebral surfaces and the sinal surface, tegman mastoidea and zygomatic dura, removed the causative focal factor in this case. Thereafter we dealt with tissue changes which were the result of an invasion from these sources. The repeated transfusions kept the chemical cerebrospinal reactions so far toward

the normal that bacteria could not seat themselves and establish an actual tissue infection. I believe, nevertheless, that the tissues at the base of the skull were edematous and pressed upon the sixth nerve, causing the diplopia, and the very edematous thickening of the meninges in some unknown way impeded the circulation of the cerebrospinal fluid and produced a stasis which distended the ventricles. To an extent, this is hypothetical reasoning, but because the girl's full recovery occurred, I was spared an autopsy from which actual clarifying data might have been obtained. Here recovery amply compensates for the loss of more accurate deductions.

The last case I will sketch for you is another type of postmeningitis otogenic hydrocephalus. In this case, for some unexplained reason, the onset of the hydrocephalitic lesion was heralded by symptoms strongly suggestive of a temporosphenoidal (Sylvian fissure, Broca's convolution, Isle of Riel) lesion. X-rays taken on admission showed a slight breaking down of the mastoid, and because of the elevated temperature and pulsating middle ear discharge a mastoidectomy was performed. The postoperative course was stormy, the temperature reaching 106 F. The severe headache persisted, but a neurological examination on the sixth day was negative. Sulfanilamide had been given and transfusions were ordered. The symptoms, however, continued, and a spinal tap showed increased pressure. The child was sent to the operating room on the 10th day and an exploration of the mastoid made. No fistula could be found into the petrous pyramid. The dura was elevated, but no extradural abscess was present. All granulations and detritus were removed, the inner table completely exposed and the tegmen tympani removed. On the following day the patient was neurologically negative except for slight nuchal rigidity and a beginning Kernig. Successive spinal taps showed a continuous rise in the cell count. The patient became incontinent and occasionally irrational. The temperature fluctuated between 98.6 and 103.6 degrees. She was continued on repeated small blood transfusions, the administration of sulfanilamide and spinal taps without much change in condition. On the 10th day following the exploration, the patient developed a total spastic paralysis of the right side, facial weakness and had positive Babinski. She did not respond to questioning, and four days later did not hear or understand the spoken language and could not utter any articulate sounds. Ventriculograms made at this time showed a moderate internal hydrocephalus. Spinal taps were performed daily and the patient continued under the combined chemo- and hemotherapy. The clinical picture varied, some days the child appearing brighter and on others she was very drowsy and apparently much worse. She slept inces-

santly and responded poorly. This continued for more than one month, and then the temperature was gradually lowered and finally reached normal. The child appeared brighter and she began to understand questioning, although she could not speak at all. Treatment was continued and the patient discharged on the 83rd day following admission, with her speech greatly improved. There was still a partial right facial palsy and hemiplegia, but beginning motor activity was noted.

In this case the bone lesion was entirely confined to the mastoid process. There is ample evidence that the petrosal pyramids were uninvolved. The surgery employed was first a simple mastoidectomy, and later when streptococci were found in the fluid and the clinical symptoms gave evidence of a streptococcus meningitis, the radical mastoidectomy was performed and the entire tegmen mastoid, tegmen tympani, the inner table uncovered, the cerebellar and cerebral dura and Trautman's triangle dura were all uncovered. The bone covering the lateral sinus and the superficial petrosal veins was removed. Then, no further evidence of bone foci being found, treatment was continued by repeated spinal taps, the administration of sulfanilamide and the administration of small blood transfusions. When the facial paralysis, loss of speech and hemiplegia developed, the question of a brain abscess of course required differentiation from a hydrocephalus. The ventricles were opened by trephine, oxygen injected and x-ray films taken which clarified the diagnosis and established the fact that we were dealing with a postmeningitic hydrocephalus. Thereafter repeated taps, sometimes twice daily, kept the cerebrospinal pressure within bounds and eventually cured the case. The child made sufficient progress since her discharge from the hospital to attend a girl's camp in July, and by now has completely recovered except for a spastic contraction of her thumb and little finger of the right hand.

CONCLUSIONS

1. In conclusion, I would say that the surgical drainage of the meninges does not answer the problem presented by mastoiditis, because the lesion is usually a multiple one consisting of many foci of active infection spreading along the route of many pial vessels. The problem is more concerned with keeping the brain tissue alive. To accomplish this, results of the brain-tissue-cell activity must be removed from the region of the brain cells, and the desired means of doing this is in keeping the cerebrospinal fluid circulating. Any procedure which stops the circulation of the fluid defeats the objectives which are in view. Brain cells naturally function best and their deleterious products are removed best when they are surrounded by

cerebrospinal fluid whose chemistry is maintained as nearly normal as possible. The repeated small blood transfusion has in my hands accomplished this purpose better than any other method, and in all the cases which I have here sketched, stoppage has not occurred, and I think this is due to the prompt institution of transfusion with the first advent of the case in my care, and the continuous use of this means, irrespective of other therapy.

2. Study of healed cases from literature showed that cured cases seemed to be obtained where the clinical picture is not so stormy and the symptom complex may be described as weak, and where pressure signs predominate over signs of toxicity. Repeated taps with a study of the pressure each time helps to control this pressure by giving an intimation of how promptly the pressure falls and how long it remains low.

3. The removal of the surgical bone focus cannot be too strongly stressed. In exposing the dura surrounding the temporal bone the procedure to do this breaks the contiguity of the penetrating skeletal dural vessels and interrupts the progress along these vessels of an infection from the bone to the dura, and helps healing the meningeal lesion.

In the examination of the cerebrospinal fluid, the following factors all need evaluation in summing up a clinical picture; the physical characteristics of the fluid; the pressure under which it is obtained; its biochemistry; its cytology; and finally its bacteriology.

4. Chemotherapy, especially sulfanilamide, has given me better results than any I have obtained by any other drug, sera or vaccine. It is demonstrable in the blood and in the cerebrospinal fluid when given by mouth. Combined with small transfusions it is the best means to date which I have employed.

In giving sulfanilamide it is well to make periodic examinations to watch for the formation of metahemoglobin, a pathological by-product sometimes found in the administration of this drug. The formation of metahemoglobin must be avoided.* Its marked presence constitutes a danger sign indicating stoppage of the drug. The simultaneous administration of the transfusions helps to hold this by-

*At the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, January 26, 1938, the intravenous injection of 1/10 cc. of a 1 per cent solution of methylene blue per kilo. of body weight was reported as a catalyzing agent which hindered the formation of metahemoglobin and permitted the use of larger dosage of sulphanilamide without bad blood effects.

product within bounds and enables us to continue it longer than otherwise.

5. Rosenthal[6,7] found sulfanilamide a potent therapeutic agent in experimental pneumococcal infection with virulent strains, in mice, rabbits and particularly in rats. The curative action was most evident with Type III pneumococcal infections. Rosenthal concluded that "up to the present time no other compounds have been found as effective against pneumococcic infections as sulfanilamide."

Gross and Cooper[8] also obtained favorable results with the use of sulfanilamide in Type III pneumococcal infections in rats.

Long and Bliss[9] reported that, "our experience in the treatment of clinical pneumococcal infections in human beings has been primarily limited to those of the middle ear and mastoid. Our observations tend to show that sulfanilamide has about one-half the chemotherapeutic effect in pneumococcal infections that it has in hemolytic streptococcal infections of the middle ear and mastoid."

Mertins and Mertins[10] recently reported a case of unclassified Type IV meningitis of otitic origin which recovered following sulfanilamide therapy.

The mode of action of sulfanilamide is still mooted. Rosenthal[11] believes that "the bacteriostatic and bactericidal action of sulphonamide on pneumococci in vitro is adequate to explain its chemotherapeutic effect in animals. The nature of this action in vitro is unusual in that the drug is not an antiseptic in the usual sense." Long and Bliss, however, hold that a change takes place in the organism which renders them susceptible to phagocytosis, but the nature of this change is still unknown.

6. When a meningitis has run its course, and the fluid tends to become normal, and the symptoms present themselves suggestive of a brain abscess, it is well to perform ventriculography before accepting a diagnosis of brain abscess. If the fluid can be kept circulating, and the pressure kept down, otogenic hydrocephalus has a tendency to heal, as the three cases cited by me all tend to demonstrate.

7. Handled in this logical way, we are in a position to say, that otogenic streptococcic meningitis is on the way toward coming under therapeutic control, and the prognosis heretofore invariably grave, gives better promise now than formerly that more recoveries will be accomplished.

71 EAST 80TH STREET.

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IX

A REVIEW OF THREE HUNDRED AND THREE CASES OF CHOLESTEATOMA*

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A few months ago I saw a case of primary cholesteatoma of the temporal bone. It occurred in a 32-year-old man who, until five years ago, had had absolutely no history of ear disease. At that time he had an insidious onset of discharge. Developing symptoms of pain, nausea and vomiting, he sought relief.

Examination showed foul discharge coming from an attic perforation. In the non-discharging ear the drum appeared to be normal. The hearing tests showed middle ear deafness in the affected ear, but normal hearing in the other. X-rays revealed unquestionable bilateral cholesteatoma. Here was a man who had bilateral cholesteatoma diagnosed by x-ray, with one ear giving symptoms for five years and the other ear giving no symptoms at all and appearing perfectly normal. Operation on the affected ear revealed a large cavity filled with cholesteatoma. The other ear, because of lack of symptoms and clinical signs, was not operated upon; but because of the definite x-ray evidence, there is little doubt but there is a large cholesteatoma in that ear also. We have in this unoperated ear an example of cholesteatoma developing without symptoms or signs, which, I feel, indicates that it is of primary or congenital origin. The clinically affected ear is probably primary or congenital also, being secondarily infected, as the onset of symptoms was so insidious and had no relation to an acute infection.

Secondary cholesteatoma is formed by an ingrowth of epithelium from the external surface of the drum or canal, following an acute infection in which the drum is destroyed. This is the oldest and most commonly accepted conception of the formation of cholesteatoma of the temporal bone. A great deal has been written about primary and secondary cholesteatoma. Several articles completely review the literature on the subject.[1-4, 6-9, 11]

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*Read before the Massachusetts Eye and Ear Alumni Association, Boston, November 17, 1937.

Fig. 1a. The usual x-ray position showing bilateral cholesteatoma. The left ear was apparently the normal ear. The right ear had had discharge for five years.

Fig. 1b. The frontal-occipital view. This position outlines the superior-posterior border of the petrous portion of the temporal bones and also defines clearly the antra. The marked enlargement of both antra is very well shown.

Realizing that there were but a few cases of primary cholesteatoma reported, and that it is a rare disease entity clinically, it seemed in order to review the cases of cholesteatoma at the Massachusetts Eye and Ear Infirmary. In reviewing the cases an attempt was made to classify a case as primary or secondary. This was difficult to do and one could not positively state whether a case was primary or secondary even though the history and findings definitely pointed to one or the other. This being the case, the data obtained will be presented and briefly discussed, and you may decide whether cholesteatoma of the temporal bone is primarily of congenital origin manifesting itself when there is secondary infection or discharge, or whether in the majority of cases it is secondary to infection of the middle ear.

In reviewing the hospital records, only those cases were taken in which cholesteatoma was found and mentioned at the operation. There may be some inaccuracy here, as the cholesteatomatous material was not examined microscopically in all cases, nor was enlargement of the antrum mentioned in some cases. To check this possible error, the operative findings were re-examined and it was gratifying to learn that enlargement of the antrum was mentioned in most of the cases, together with cholesteatomatous material.

The records extended over an eleven-year period, from 1925 to 1936, inclusive, and number 303 in all.

In studying the age incidence, that is, the age at which the patients were operated upon, it was observed that 169 patients or 56 per cent of all the cases were under 26 years of age, 129 or 43 per cent of all the cases were under 20 years of age, and 34 or 11 per cent were under 10 years of age. It may be readily seen from the chart that most of the cases are under 40 years of age, showing that cholesteatoma occurs mainly in the first four decades of life. This age grouping shifts rapidly and strikingly to the younger individuals when we examine the age of onset of symptoms. This onset is usually recognized by a discharging ear, ushered in with or without an acute infection.

This grouping is striking, in that 46 or 15 per cent of the cases started to have discharge from the ear under one year of age. One hundred and four or 35 per cent began to have a discharge under six years of age, while 190 or 63 per cent began their symptoms under 11 years of age. Such statistics can be used to back up either the primary or secondary cholesteatoma theories, as it is known that most cases of otitis media occur in children. Thus it could be said that discharge starting so young prevented pneumatization of

AGE INCIDENCE

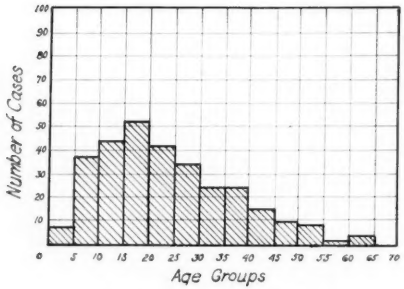

Chart 1. Explanation in text.

AGE OF ONSET

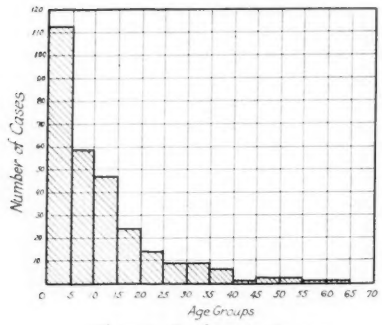

Chart 2. Explanation in text.

DURATION OF DISCHARGE

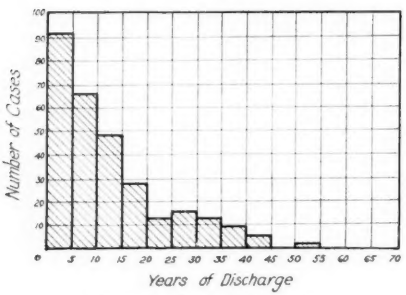

Chart 3. Explanation in text.

the mastoid, permitted the ingrowth of epithelium from the auditory meatus and started cholesteatoma formation, thus favoring the secondary school. On the contrary, if cholesteatoma is present at birth, as primary cholesteatoma is, any infection that occurs would tend to persist and the cholesteatoma would be found at operation. Here then is evidence in support of both factions.

The duration of the discharge in this series seems to favor the conception of primary cholesteatoma, because in the majority of cases the length of discharge is comparatively brief, too brief for cholesteatoma to develop from the epithelial invasion from the canal and form the large cavities that were found at operation.

Fifty-nine cases or 20 per cent had discharge lasting less than one year from the onset and 202 cases or slightly over two-thirds had discharge less than six years. Of course, there are some that had discharge for over 40 years, but these were in the minority. To discard this evidence in favor of the primary school, Nager[1] presented some very valuable observations. He studied several cases of fulminating scarlet fever. He saw the intact drums before perforation, saw the drums melt away during the course of the disease and then was able to examine the temporal bones microscopically at post mortem. Examining these specimens, he observed the epithelium growing into the middle ear from the drum margin. His cases showed this ingrowth of epithelium to be occurring within 20 to 45 days from the onset of the infection. With this evidence one is impressed by the rapidity with which epithelium can invade the middle ear.

Other observations are the locations of the perforations in the drum. In 34 cases the perforation was recorded as in Shrapnell's membrane, in 32 cases the perforation was recorded as being posterior, in 18 cases it was anterior, in 18 others it was central, in 48 the perforation involved the entire drum, and in one case the drum was intact and normal.

In certain cases the drum could not be seen, as it was hidden by polyps or granulations. Polyps were observed in 64 cases, granulations were observed in 59 cases, and cholesteatoma was mentioned as being in the middle ear in 33 cases. The outstanding observations here are that the majority of perforations occurred where one would expect to find them in cases of secondary cholesteatoma; that is, in the posterior or superior part of the drum. There were also a large number of cases that did not have perforations so located, but which were located in the anterior or central part of the drum.

Perforations located in the posterior or superior part of the drum are expected and accepted by the secondary cholesteatoma school, but the perforations not located there are more difficult to correlate with the theory of secondary invasion, and rather favor the presence of cholesteatoma before the perforation occurred.

A strong argument in favor of the secondary cholesteatoma was found in 25 cases that were operated on a second time, in which no cholesteatoma was found at the first operation. The time elapsing between operations varied from one month to twenty-two years, while the age varied from three and one-half to forty-eight years. The age of the patient and the time interval between the first and second operations had no correlation, although all of the patients but three were under 26 years of age when the first operation was performed and one-half of these were under 15. Certain of these cases must have had some cholesteatoma present in the attic or antrum that was not seen or mentioned at the first operation, or else the finding of foul débris at the second operation gave the impression of cholesteatoma and was so reported by the House Officer. As an example of this, there was a patient, 39 years old, who had had a discharging ear for seven years and cholesteatoma was not found at the first operation, and yet one month later at a second operation it was observed and recorded.

The x-ray diagnosis of cholesteatoma has become much more positive during the past few years in that all patients who have mastoid x-rays taken, now have the fronto-occipital position included. This position shows up enlargement of the antrum more readily than other positions. Making no allowances for this improvement in x-ray technique, the x-ray interpretation was nevertheless very accurate in demonstrating cholesteatoma. One hundred and sixty-five cases out of this entire series had enlargement of the antrum and all of them had cholesteatoma at operation. Of the cases not diagnosed by x-ray, the x-ray report was "complete sclerosis" in 95, "post-operative" in 29 and "some destruction" in 14.

Now that my evidence is presented, permit me to correlate my findings with the statements of a few of the writers on the subject. Nager[1] stated that in over 100,000 cases of cholesteatoma none gave positive evidence of primary origin. This series of 303 cases that I have just reviewed does not agree with Nager, in that there is one case of primary cholesteatoma proven by x-ray and substantiated by operative evidence of cholesteatoma in the other ear. In this same series 20 per cent of the cases had discharge less than one year and nine had discharge less than three weeks, which would indicate

Fig. 2. A picure of x-ray position used to demonstrate cholesteatoma and petrous apex.

that the cholesteatoma was present before the infection started. Then there is the age group of 190 or 63 per cent of the cases that were 10 years or under, suggesting that the condition may have been primary since infancy. These three observations point to the fact that primary cholesteatoma does occur.

In another series of observations Ulrich[5] pointed out that cholesteatomatous ears could not have central perforations. This was based on 458 operations, 98 per cent of which had epitympanic perforations. Again this small series that we have reviewed does not agree, as 18 cases had anterior perforations and 18 others had central perforations.

Ritchie Rodger[1] presents an argument that cholesteatoma is secondary in origin. He states that cholesteatoma are frequently bilateral, as are many cases of otitis media, and yet bilateral new growths are almost unknown. This may be true, but, if cholesteatoma is considered as arising from congenital enclosure of epidermal cells, it seems very logical that they should be bilateral, as are cysts in front of the tragus due to incomplete closure of the first branchial cleft.

Almour,[10] in his discussion, cannot explain how a pneumatized mastoid could become sclerotic without the mechanism of Wittmaack's theory, and yet such cases do occur. Dr. MacMillan has presented a series of x-rays which show a pneumatized mastoid going through the various stages of infection and healing completely sclerosed.

Another point which is in favor of the secondary cholesteatoma is that in this entire series of 303 cases there is only one that shows x-ray evidence of cholesteatoma without clinical evidence of its presence. Add to this number 14,000 other mastoid x-rays that were taken during the same period, none of which showed evidence of cholesteatoma, and one is impressed by the fact that there are very few cases of primary cholesteatoma that do not have symptoms that arise early in life.

SUMMARY

Evidence in Favor of the Primary School

One case of primary cholesteatoma proven by x-rays.

The age of onset in two-thirds of the cases was under 11 years, and in 15 per cent of the cases it was under one year.

The duration of discharge was brief before operation. Twenty per cent of the cases had discharge less than one year and nine had discharge less than three weeks.

Location of perforation

18 were anterior.
18 were central.
1 the drum was intact.

Evidence in Favor of the Secondary School

Twenty-five cases in which no cholesteatoma was present at a first operation, but was found at a second operation.

One-third of the cases were over 11 years and eight cases started symptoms after 40 years of age.

One-sixth of all the cases had discharge lasting more than 20 years.

Location of perforation

34 were in Shrapnell's.
32 were posterior.
48 the drum was gone.

We now have the recorded facts before us and the theories of primary and secondary cholesteatoma both appear to have points in their favor. Without question both types do occur, as there has been presented in the literature evidence that shows probable beginning cholesteatoma in the foetus without signs of infection[11] and also cholesteatoma developing in a normal ear after an acute in-

fection.[4] Is it primarily of congenital origin manifesting itself when there is secondary infection or discharge, or is it secondary to infection of the middle ear with resultant cholesteatoma formation? I personally believe that both types do occur. I also believe that the evidence presented in favor of primary cholesteatoma, backed by an impression gained by a review of the literature, justifies the statement that primary cholesteatoma is present much more frequently than most of us have believed.

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X

PROBLEMS IN DIAGNOSIS AND TREATMENT OF HYPERPLASTIC SINUSITIS AND ALLERGY*

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In the practice of medicine or surgery today one is cognizant of an ever widening horizon. The range of knowledge continues to expand and the narrow realm of the known such as prevailed in past times can no longer be mastered by a single individual mind. Hence, we find it imperative for men to limit their activities to special regions of the body to attain any degree of perfection in the science of their profession. A more comprehensive knowledge, on the other hand, tends to enlarge the field of each specialty, at the same time to make it less of a specialty and to demand on the part of all specialists a more intimate knowledge of the correlation of the phenomena which have a local habitation and a name with various processes of a nature known or hypothesized in other parts of the body.

Of all such pathological processes which influence the function of individual organs and show its manifestations in a variety of tissues and locations, none is more observed and studied than allergy. It is so protean in its aspects that it invades the realm of almost every specialist, and no specialist can afford to be delinquent in his attempts to acquire some knowledge of its etiology, diagnosis and treatment. Thus the surgeon must be mindful of its capricious deceptive capers in the abdomen and should give such a possible cause of symptoms some thought before a laparotomy is performed. The orthopedic specialist likewise should be suspicious of some of his joint conditions as an allergic phenomena, and beyond all others the rhinologist may be suspicious of all his cases which manifest any evidence of nasal obstruction. The latter will not usually lack sufficient warning from the allergist if he shows a propensity to operate on noses that are possibly allergic. In fact, if the rhinologist reads too many books on allergy, he may develop a fear complex toward ever doing any nasal surgery.

I do not wish by such a statement to disparage the work of allergists, for their discoveries have produced one of the great quiet

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revolutions in the practice of medicine, almost as epochal as the germ theory of disease, and we acknowledge with gratitude their great help in the solution of many of our problems.

Let us now consider some of the many pathological processes in the nasal cavities and paranasal sinuses where the allergist and rhinologist must cooperate to insure proper diagnosis and treatment. The seasonal hay fever cases will present slight difficulty in diagnosis and are now generally treated by about the same methods, either by specific avoidance of the irritant or by desensitization with standardized extracts. If symptoms prevail in these cases beyond the hay fever season, one must always be suspicious of engrafted sinus infection. Most of them tend to get well, however, as the allergy subsides, and the nose acquires thereby better ventilation and drainage. A little judicious intranasal treatment will often facilitate this. If, in spite of this, symptoms prevail, it is wise to test the patient for other allergic factors such as house dust, orris root and feathers.

The sporadic cases with symptoms like acute nasal allergy often presents considerable difficulty in diagnosis. Such cases present themselves at any season of the year, and while they may be a true allergy, there are a great many which are not. A careful nasal examination may reveal a purely local cause for such attacks. If occurring after a coryza one may be suspicious of retention of pus in one or more ethmoid cells. They should be treated by a thorough shrinking of the upper part of the nose with ephedrine 3%, or neosynephrin 1%. A suction irrigation of warm saline used afterwards will often dislodge several casts of ethmoid cells and the symptoms will clear up rapidly.

Other cases may be due to points of near contact in the nose, between the middle turbinate and bony spurs of deflections. These patients may have no symptoms until slight swelling of the membrane occurs, and then the tickling sensation begins. The swelling may be caused by entering a warm room or even the slight nasal congestion associated with an overloaded intestine, mild indulgence in alcohol and numerous other factors. The areas of contact which produce these symptoms later on with the membrane becoming more thickened, often give rise to constant headache. A single application of cocain will relieve most of these patients for some time, but a permanent cure is effected by either removing a portion of the middle turbinate or by a submucous resection of the septum, or both. I can recall many cases of this nature which had been previously diagnosed as allergic.

The true allergic coryza may be due to various inhalants, foods or drugs. The inhalants most common are house or occupational dusts, orris root, animal emanations and feathers. Diagnosis is determined by the historical method and sensitization tests, either on the skin or mucous membranes. In routine practice it is surprising how many times a cause can be determined or suspected by asking a few leading questions. For example, a patient came to me saying she had something like a cold in her head for three weeks, though it did not come on like a cold. I looked at her nose and found the membrane swollen and some mucoid watery secretion. I found she had not changed her environment in any way. I asked her if she had had the same kind of food as usual, and on replying in the affirmative, I asked her if she was certain that there had been no change. She laughed and said everything was just about the same except something too silly to mention, "Of late we discovered a new type of spinach which we like very much and have eaten it a great deal." I sent her home, sworn to total abstinence from the spinach, and in a day or two her nose was normal. A history, of course, has usually to be more elaborate than the above, and the patient's occupation should always be considered first.

It always seems advisable to take a preliminary history, then make an examination of the nose. If, after this, one has a suspicion of allergy, a more complete history should be taken. If this provides more evidence in favor of allergy, a series of elimination tests can be tried and results carefully watched. Failing in this method, skin tests may be found useful, but are not always reliable, since many positive reactions occur in normal persons and a considerable number of allergic cases do not show positive skin tests. Pottenger[1] has noted a different skin reaction in bacterial allergy from nonbacterial sensitizing agents. Bacterial allergy, the positive skin reaction does not show itself until about 24 hours, and is popular in character rather than the characteristic wheal of the nonbacterial type, though the former may occasionally show an immediate reaction.

The conditions which give the most trouble in diagnosis are those where clinical evidence is not certain as to the presence of allergy, and other obscure causes may be operating to produce symptoms. In some cases a very thorough family history may help us, as the hereditary factor is most important. Various observers have shown a percentage varying from 25 to 30 per cent of persons showing some manifestations of allergy in family groups.

The protean nature of the disease must, of course, be remembered in taking the family history and inquiry made about the incidence

of asthma, hives, eczema, hay fever, angio-neurotic edema, migraine, gastro-intestinal disturbances and allergic rhinitis. Some may suffer from one or several of these known allergic diseases. Also, in the individual history, one should look for manifestations in other parts which might be correlated to the symptoms under investigation. The various family and individual idiosyncrasies to food or environment should be determined, although the common plea that certain foods disagree with people may or may not mean that they are allergic to such foods. As a matter of fact, the foods producing the worst allergic symptoms are often those particularly relished and are not provocative of any gastric or intestinal symptoms of which the patient is conscious. There are, however, exceptions where the immediate ingestion produces marked gastric symptoms. As a rule it would seem that where a food excites pronounced indigestion the secondary symptoms of allergy are not apt to occur.

If we now assume from picking up a definite clue from the history that we are on the proper trail of an allergic symptom complex in our own field, we should inquire about the time of day when symptoms are most aggravated. Nearly all allergic symptoms are worse at night and some produce this discomfort almost entirely in that period. With all this preliminary data we may now proceed to acquire our objective evidence, namely, by skin or mucous membrane tests, as advised by Dean.[2] Examination of smears from the nose for the number of eosinophils and their numerical percentage in the blood should also be determined. The nasal smear will likely show a far greater number of eosinophils in allergy than will be found in a smear from an infective process. The finding in the blood is not pathognomonic, but eosinophilia varying from 5 to 20 per cent is often present. Kahn and Stout[3] consider that a nasal smear with 10 to 90 per cent eosinophils is positively diagnostic of nasal allergy, while 4 per cent or over is suspicious. In infectious processes the polymorphonuclear leucocytes are much more abundant. Bray[1] and other observers have reported a hypochlorhydria as prevalent in allergic cases.

Let us now assume that we have a patient with a positive allergic history and that all tests are positive, we then proceed with our complete nasal examination. In mild cases there will be the usual signs of enlarged turbinates and general nasal obstruction, the condition described in older textbooks of hypertrophic rhinitis. If the nose is wet and subject to frequent coryza attacks, with sneezing and free watery discharge, we would be well advised to refrain from all treatment except the usual palliative measures. The nature of the allergy in such cases must be determined and must guide the treatment. Even

though the nose shows marked anatomical defects they had better be left alone until the allergy is brought under control: Even a simple submucous in such a nose will be followed by a very protracted convalescence, with an unsatisfactory result. There is, however, a type of case much more chronic in nature with enlarged turbinates and very little discharge. These patients often complain of very frequent exacerbations when they think they are taking a fresh cold. The allergy in such cases should always be treated, but one can often do a great deal for them by minor surgical procedures, undertaken, of course, during a quiescent period. A submucous may be done, usually supplemented by cauterization or scarification of the inferior turbinates and perhaps a partial removal of the middle turbinate on one or both sides. Such cases will often get such a satisfactory result that one may afterwards have doubts as to whether they were allergic after all. It is in this type of case that a vicious circle has been established by the nasal obstruction *per se* with its areas of contact as mentioned above. Even though tests for allergy were positive there is still a legitimate reason for believing that it is not the important factor. Cooke[5] does not believe that substances producing nasal symptoms from ingestion are very common, estimating not more than two or three per cent. This is probably correct, for the more chronic type of hyperplastic nasal pathology, but many periodic short attacks of allergic rhinitis can be traced to something in the patient's diet.

The next type of case to consider is the more pronounced hyperplastic type with polyp formation, in which the history and tests are positive for allergy, though there are no active manifestations of allergic phenomena. Here again if there is an obvious allergic factor it should be treated and if not too far advanced, and of long standing, there may be sufficient recession of the polyps to relieve the local symptoms. Failing in this the polyps should be surgically removed as thoroughly as possible. These cases will need very careful after care and any recurring polyps should be removed at once. In spite of this, unless the allergy is brought under control, there will be some recurrence. The question whether all polyp formations are associated with allergy is much debated. In a series of polypi cases observed by Kern and Schenck[6] it is found that all were allergic or showed some allergic tendency. Cooke[7] describes the cause of hyperplastic conditions with or without polypi to a primary infection in a person with an allergic constitution and does not believe that specific treatment of the allergy will produce any change in the hyperplasia. He believes the allergy is a sensitization to the infection. It is hard to deny such a supposition, but clinically we do find a severe

ethmoid infection with necrosis of intercellular septum, exuberant granulation, pus and polyps which show no general evidence of being allergic. In examining these cases we find perhaps only a history of a severe acute infection as the beginning of trouble. Their nasal mucous membranes show no suggestion of allergy, especially the membrane covering the nasal septum. Whereas this will be involved in the allergic cases, even polyps may form on the septum in the latter type of case. A smear of the nasal secretion will show the preponderance of polymorphonuclear cells, also the trouble is often localized in the beginning in one side of the nose or one group of cells. They do not tend to recur after removal to the same extent as the allergic cases. I can recall many cases where there has been no sign of recurrence over a period of ten to fifteen years. Another reason for believing that such polypoid cases are not necessarily allergic is the fact that they have been totally relieved of symptoms and permanently cured by surgical methods. This, of course, does not prove that they were not sensitive to their bacterial proteins which disappeared with the eradication of the focus of infection, but many present only symptoms of a chronic infection.

It is very common in the routine of rhinological practice to find the two factors on allergic diathesis with a superimposed infectious process. Both factors should be treated, and it is just as well to give the allergist a chance to do what he can before any attempt at surgery is undertaken. If bacterial allergy is suspected, a long course of vaccine treatment may be worth trying. I think the only result I have ever seen with vaccine in sinusitis is one of desensitization. This does not cure the sinusitis but may afford relief from symptoms.

The association of asthma with any of the above type of cases has led to a great deal of discussion. The majority of allergists believe that the intranasal polyp formation is merely an association and not related as cause and effect. They are inclined to condemn the futile attempts to cure asthma by intranasal surgery. There is also a great discrepancy in reported results by rhinologists. This can be partially explained by the variable quality of intranasal sinus surgery, but even in the most skillful hands there is a large percentage which fail to improve.

The earlier the cases are seen after the onset of asthma the better the prospect for the final result. I have seen a submucous resection of the septum effect a permanent cure in one case where this was done shortly after the onset. Another was almost completely relieved by a similar procedure, to give ventilation and drainage of an infected ethmoid. I am certain that I have never done radical surgery in a

nose with polypoid degeneration associated with asthma in which I did not get some improvement, with an occasional cure. Since these cases are so miserable from the local discomfort of a blocked-up nose, I think one is justified in clearing them out. Though one can never promise very much about curing the asthma, I don't know of anything else that offers even as much promise as surgery in obstinate cases. Two recent reports quoted in the 1937 Year Book show the usual variation in final results from intranasal surgery. Dr. E. DeStio's[8] series of thirteen cases show three cured, three improved, with no result in seven. Fox and Larned[9] from a study of 150 cases of asthma, forty of which had sinus operations performed, report cured in from 45 to 60%, depending on the type of operation, the more extensive operations as usual producing the highest percentage of results. The lapse of time subsequent to the operative work should be considered, as many will only begin to show improvement after six month or a year. Gradual improvement may continue till the disease seems to wear out, with only slight flare-ups during an acute reinfection of the sinuses.

The other cases in this series were treated by various methods with results less favorable than the surgical cases. Of late years there have been many reports of treatment of nasal allergy by ionization, with as much discrepancy in results as in surgical methods. Alden[10] and Tobey[11] have both reported favorable results. MacMahon[12] has shown experimentally that ionization is a destructive process, producing marked fibrosis of the membrane with decreased ciliary action. Dean's[13] conclusion in regard to ionization is that it produces such deleterious results on the mucous membrane that the ill effect outweighs the good. Application of pure phenol to the nasal mucous membrane has been tried and good results reported, but as this also acts by destruction of functioning elements of the mucous membrane it does not recommend itself as a method of treatment.

The application of silver nitrate in strength from 5 to 10% often gives satisfactory results. It is surprising how even polypoid areas will shrink with such treatment. X-ray treatment has been advocated and practiced and good results reported. I have had no experience with it, but it seems reasonable to suppose that it might be beneficial if carefully administered. I have seen very little permanent result from diathermy in polypoid cases.

Other combinations of nasal allergy with infection may be the possible explanation of some types of headache. Apart from migraine there are many cases of headaches caused either by local swelling of the mucous membrane in the nose or sinuses or possibly from cerebral

edema. The headache of the "morning after" may be of this type; other forms may be periodic with associated nasal symptoms, or a steady type with slight remission. This latter variety of headache can be very severe and disabling. The pathology found is a thickened membrane within the ethmoids and sphenoids, even completely polypoid with no sign of polypi in the nasal cavities and either no discharge or a small amount of postnasal mucoid drip. It would seem that polypi within the cells can exercise enough pressure to give pain, whereas polypi that have broken through the cell walls and filled up the nasal cavities are very seldom associated with any pain. The x-ray will show this condition very clearly, especially one taken in the vertico-mental position, and is better evidence than one can obtain from the most careful clinical examination. In fact some cases examined after cocaine application will show practically nothing. Patients will complain of some nasal obstruction worse during the pain exacerbations and perhaps of some postnasal discharge. The allergy usually seems to be the dominant factor, though it is probable that infection was the initial cause. When the intracellular membrane has undergone such a change anti-allergic treatment will give no respite, and surgery offers the only promise of relief. A thorough exenteration of the ethmoids and making large openings into sphenoids is necessary. The after care must be carried on for some time as the sphenoid openings in these cases have a great tendency to close; if allowed to do so headaches will return. The pathology as described is very obvious at the time of operation, if done under local anesthesia one may see the polypoid tissue as the cell is opened. Under general anesthesia the ethmoid labyrinth has a feeling of doughy consistency with irregular areas of resistance due to absorption of intercellular septa. I have operated on a good many cases of this kind after they had been treated by excellent physicians for years with no success. The headaches ceased in one patient immediately after operation; others have obtained great relief, and I cannot recall any who would regard the operation as a complete failure. Probably in these cases, as in asthma, one will find a great discrepancy in reported results. I cannot refrain from making a cynical observation about some of the ultra-conservative opinions concerning sinus surgery. Honest statisticians are not always good surgeons, and from a long experience with secondary sinus operations I feel that there are very many of them badly done. Incomplete operations may do more harm than good and merely stir up an infectious process without removing it. On the other hand some of the over-optimistic reports would suggest that the operator is drawing upon his imagination for his facts.

In a brief review such as this of a very difficult problem, it is hard to even add a grain of truth to what is already known. The expression of individual opinions, based on long experience, in the course of time helps us to formulate some standards of right thinking in the problems of diagnosis and treatment. Allergy is a wide field, and looking at it from a biological viewpoint it seems to be a failure of adaptation to some of the minor shocks of our environment, a derangement of the automatic switch board which regulates the balance of the autonomic nervous system. The constitutional vulnerability may be induced by some infection in early life or some sudden or prolonged emotional strain with heredity being a very potent predisposing factor. The exciting causes include physical and chemical agents either from without or from disordered biochemical processes within the body. So the study of each individual case demands a comprehensive understanding of the whole science of medicine.

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XI

A MASKER FOR BONE CONDUCTION TESTS

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In cases of unilateral deafness the deaf ear registers a spurious perception of sound whenever the stimulus acting on the defective organ has large enough intensity to cause acoustic vibrations of skull tissues and of the opposite labyrinth. In audiometric testing the "shadow-curves" obscure the true status of the hard of hearing ear. When the air conduction curve of the deaf side is 25 to 40 sensation units below that of the normal side we may suspect in the worse ear a defect much larger than the shadow-curve indicates. Shadow-curves obtained in tests with bone-conduction receivers are still more misleading. They are usually only 10 to 15 sensation units below those of the good ear, but the difference may be much smaller in one or more frequencies. The subject becomes still more complicated if both ears are affected with different degrees of hardness of hearing.

The true bone-conduction curve may aid the diagnosis and is certainly of utmost interest to the students of physiology and pathology of hearing. For the purpose of eliminating the function of one ear the method of "masking" is generally accepted. It consists of "deafening" one ear with a sufficient amount of noise while the other ear is tested with tones of definite frequencies and intensities. The original instrument made for this purpose is Bárány's "lärmapparat." The device, however, is objectionable because it emits too much extraneous noise and so masks not only one, but both ears.

The phenomena of masking were studied by Harvey Fletcher[1]. His researches and theories on this subject can be shortly summarized as follows: If a pure deep tone (i. e., below 500 double vibrations per second) acts on a drum-membrane, the simple regular vibrations, provided they reach certain intensities, are transformed by the middle-ear mechanism into complex regular waves. The cochlea then receives vibrations containing subjective tones—harmonics.

The harmonics, being of higher pitch than the basal tone, have a similar masking effect on the high frequencies as has the basal tone on the low frequencies. The intensity of harmonics and consequently

also the masking effect of the pure deep tone decrease with the pitch of the harmonics. High pitched pure tones mask only perception of high tones and do not affect the low tones. Simultaneous masking by two or more tones results in summation of masking effects of the components.

Fletcher's analysis confirms Bárány's contention that hearing can best be eliminated from one ear by a mixture of tones, i. e., by noise. The problem is to produce a noise which (1), consists of proper mixture of pitches and intensities, and (2), reaches practically only one ear, leaving the opposite ear unencumbered for delicate hearing tests.

The American Otologic Society[2] recommended three devices for masking: (1), The telephone buzzer; (2), The electric door-buzzer, and (3), Current of air blown into the meatus. Each of them has serious defects. The telephone buzzer, if used with air-conduction receiver, does not sufficiently mask the tones above 2,000 double vibrations and emits too much extraneous noise; in bone-conduction tests it does not sufficiently eliminate the hearing of the good (masked) ear in speech frequencies (256, 512, 1024), and yet it interferes with the hearing of the worse (tested) ear if its loss of hearing is less than about 40 sensation units. The masking effect of the door-buzzer is similar to that of the telephone buzzer except that the amount of extraneous noise is larger. Blowing of air against the drum-head produces very little extraneous noise and is rather satisfactory for testing with the air-conduction receiver over the opposite ear. However, in the bone-conduction tests this method of masking leaves rather high shadow-curves.

After some experimentation the author constructed a simple apparatus which produces noise by a combination of the electric door-buzzer and of the air-current expelled from a rubber balloon. The door-buzzer is enclosed into a container with acoustically insulated double walls. Its noise is conducted to the ear by a stethoscope-like device and is "mixed" in the receiver with the air-current. Tests on cases of unilateral deafness and control experiments on normal ears indicate satisfactory performance if the instrument is used for intensities and frequencies produced by the Western Electric 2B Audiometer in either the air-conduction or the bone-conduction receiver.

DESCRIPTION OF THE MASKER

The apparatus consists of three main parts. These are represented by Figures 1, 2, and 3. Fig. 1 shows the acoustically insulated

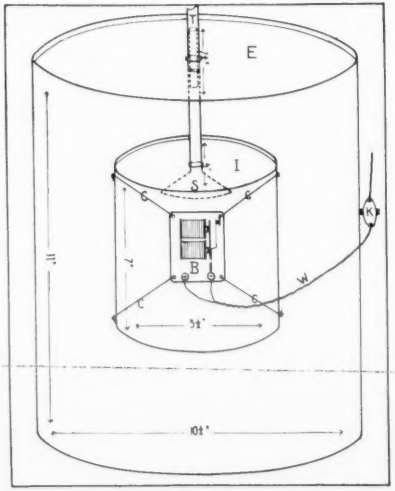

Fig. 1

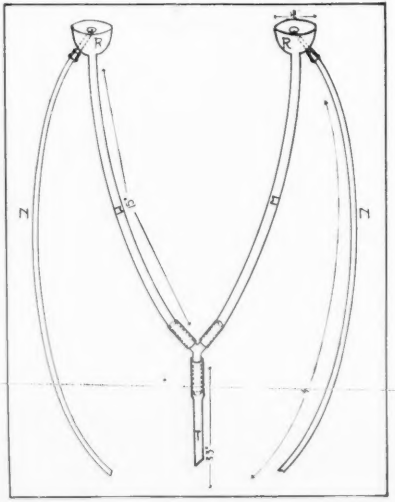

Fig. 2

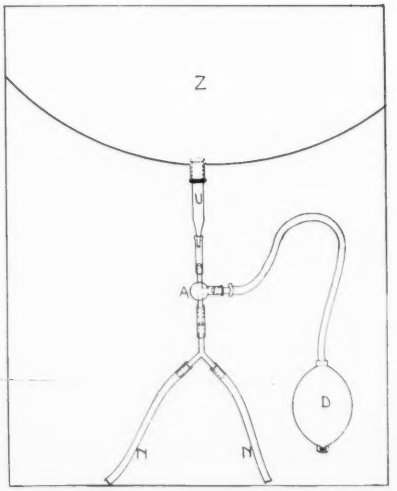

Fig. 3

Fig. 1. The enclosed buzzer. E, outer container. I, inner container. B, buzzer suspended on cords C. W, K, electric wire and switch. S, rubber funnel receiving the sound.

Fig. 2. The stethoscope. R, Receives. M, T, tubes conducting the sound from the buzzer. N, tubes conducting the air-current.

Fig. 3. Z, Rubber balloon. A, three-way switch. D, hand-bulb. U, Young's syringe. N, tubes conducting the air-current.

electric door-buzzer B. The buzzer is obtainable in hardware stores in two sizes. The smaller size is satisfactory. It is suspended by means of four strings C inside the inner card-board container I. The inner container is inserted into the outer card-board container E and the space between both is packed with soft towels. Insulation with heavier material (for instance sand) is more effective but technically more difficult in a home-made device. A rubber funnel S intercepts the noise and conducts it through a soft rubber tube T (½" in diameter, Fig. 2) to which two receivers R are attached by means of connecting tubes M. The air current comes into the receivers through two ⅛" glass pipets directed parallel with the long axes of the meati when the receivers are applied against the ear. A soft rubber tube N (diameter ⅛") connects the pipets with a metal Y tube which receives air from rubber balloon Z (Fig. 3). A three-way switch can either release or stop the air-current, or it allows refilling of the balloon by means of a hand-pump D.

The audiometer is placed on a typewriter table. The containers with the buzzer are suspended under the table on four strings. The extraneous noise is increased by resonance if the buzzer stands directly on the floor. The stethoscopic device hangs on a little bracket screwed into the edge of the table. The balloon is suspended from a wall bracket or from the ceiling, or it can rest on the extention board of the typewriter table. The buzzer is operated from a three-cell dry battery to which it is connected by a wire W with a switch K. A rheostat on the battery may be of advantage if experiments with different amounts of the noise are to be performed.

The material necessary for the construction of this masker is conspicuous by its simplicity. The cardboard containers I and E were originally cheap wastepaper baskets bought in Kresge's department store. The stethoscope device has the same shopping source: It originates from two rubber shower-hoses. The receiver R is the part that fits over the bathtub tap. The rubber funnel S is the other end of the hose—from which the sieve has been removed. The connecting rubber tube T should have a heavy soft wall in order to diminish the extraneous noise and to prevent kinking which affects the intensity of the masking sound. The three-way switch belonged originally to a blood transfusion apparatus and is obtainable in medical supply stores. The hand-pump D was taken from DeVilbis spray. The rubber balloon is a beach-ball, the larger the better. It is connected to the narrow tubing by means of a urethral syringe U.

A certain amount of "tuning" of the buzzer is necessary: The noise, when listened to through the stethoscope should resemble a

deep hum comparable to a tone in the neighborhood of C128. One or two tones higher than C128 is most satisfactory. The buzzer also emits a metallic din with a pitch between 1,000 and 2,000 double vibrations. A great part of the din is lost in the tube, but if too much of it is carried to the masked ear it penetrates through the head into the opposite ear and lowers the perception of c^{3}1024 and c^{4}2048 by five or ten sensation units. The best way to damp the metallic din is to tie a piece of rubber eraser against the metal plate of the buzzer. The rubber balloon has to be pumped up before each test to deliver sufficient flow of air.

Properly adjusted and with about five volts on the terminals of the buzzer (obtainable from three 1½-volt dry cells in series), the masker emits about 60 to 65 decibels of noise at the opening of either receiver. If the stethoscope hangs down and the observer is placed about 1 m. from the apparatus the extraneous noise is about 20 decibels when listening with both ears and about 15 decibels when listening with one ear turned away from the apparatus, the opposite ear being plugged. One receiver only is required for the clinical tests. But because the apparatus itself has to be tested and occasionally checked up, the stethoscopic arrangement was found to be preferable. By applying the noise to both ears the masker can be tested against a normal observer as described later.

TESTS FOR THE MASKING EFFECT

The masking effect of the instrument was evaluated in three tests: (1) Against a known case of complete unilateral deafness. (2) Against a case of deep congenital unilateral hearing defect. (3) Against two individuals with normal hearing.

First test: (Observer No. 1, Fig. 4.) This observer was 30 years old. When he was a boy of 13 his skull was fractured in an accident and he lost the hearing in the left ear. Caloric test of the left labyrinth was negative for hot and cold water. The right labyrinth showed normal caloric reaction. The Gellé test on the left side was negative in all the eight frequencies of the 2B audiometer. On the right side it was positive in the whole range. Otoscopic examination revealed no pathology of either ear. Both eustachian tubes were patent.

Audiograms I and II of Fig. 4 present the tests recorded under usual acoustic conditions of a quiet office (curves 1, 3, 5, 7). These are compared with the second series of curves (2, 4, 6, 8) which were registered while the masker was running about 1 m. from the ob-

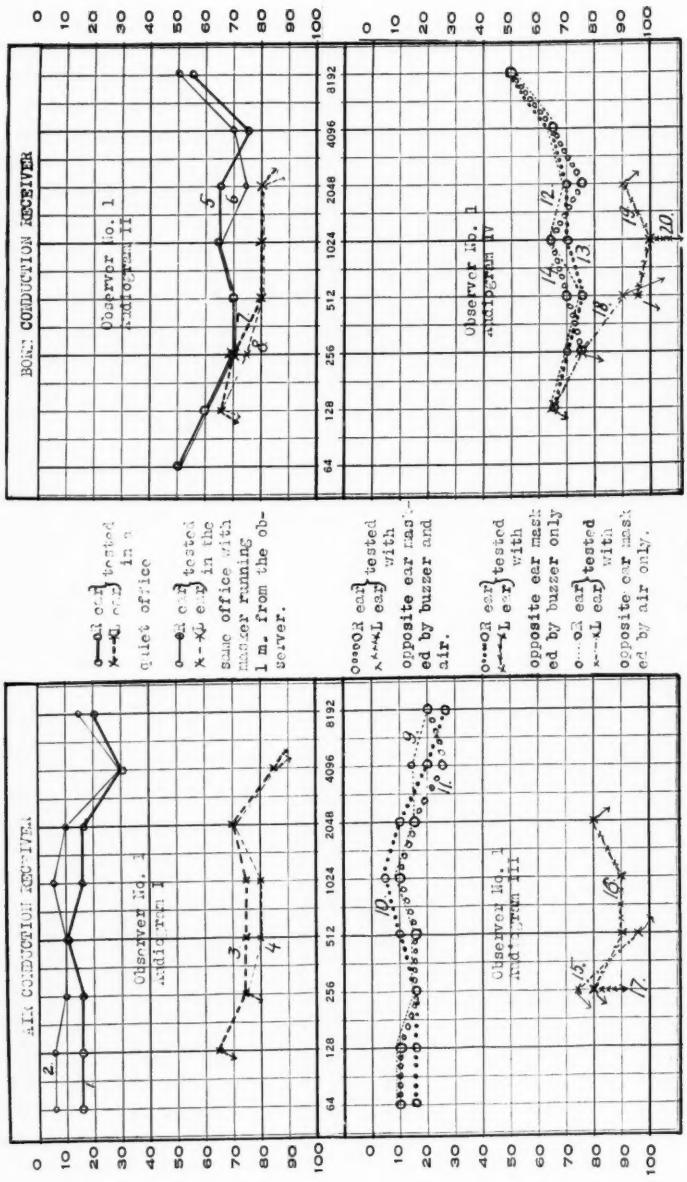

Fig. 4. Test of the masker against observer No. 1, who is affected with total traumatic deafness of the left ear.

server. As was mentioned previously, the extraneous noise of the masker equals 15 to 20 decibels under such arrangement.

Listening to the air-conduction receiver (Audiogram I) the observer registered a large difference between the acuity of his ears (curves 1 and 3). The bone-conduction curves (5 and 7) are much less separated. This observer's good ear had better meatal conduction when the 20 decibel noise was present in the room. (Curve 2 compared with curve 1.) It was a definite case of paracousis Willisii: Curves 1 and 2 were retaken on several occasions and each frequency was tested separately first without, and then immediately with the noise. The threshhold was always better with the noise. The paracousis, however, did not appear in the shadow curve of the left ear (curve 4). The extraneous noise of the masker did not interfere to any significant degree with the bone conduction tests (curves 7 and 8).

Audiograms III and IV show the effect of the masker when applied to the one or to the other ear. If the air was blown into the deaf ear (curve 9) the effect on the air-conduction acuity of the good ear was paracousis. Somewhat similar, especially in the middle ranges, was the effect of the buzzer tone applied to the deaf ear (curve 10). With both components (air and buzzer) acting on the deaf ear, the hearing of the good ear (curve 11) receded to the original level of curve 1. The same experiment was repeated with the bone-conduction receiver, but no significant changes appeared in the bone-conduction acuity of the healthy ear (Curves 12, 13, 14, Audiogram IV).

The picture was quite different when the masker was applied to the healthy ear and the audiometric receivers were listened to through the deaf ear. Here the air current alone was sufficient to extinguish almost the whole range of the meatal perception as tested with the air-conduction receiver (Curve 15). The sound of the buzzer alone was less efficient and permitted hearing if the stimulus was 80 to 100 decibels above the normal threshhold (Curve 16). However, the combination of both noises extinguished the whole range of the shadow hearing, which originally gave curve 9, and allowed only perception of 256 double vibrations raised 80 decibels above the normal threshhold (Curve 17). Repetition of the experiment with the bone-conduction receiver (Curves 18, 19, 20) showed a decidedly smaller efficiency of the air current for the bone-conduction masking (compare Curves 15 and 18). The buzzer-tone acted in the opposite sense: It masked better the bone-conduction receiver than the air-conduction receiver (compare Curves 19 and 16). Combined

noises extinguished the whole bone-conduction shadow (i. e., the curve 7) except the 1024 d.v. tone 100 decibels above the audiometric reference point (Curve 20).

Conclusion of the first test: Combined air and buzzer sound of the described masker completely extinguished the air-conduction and bone-conduction shadow curve in a case of known unilateral total deafness. This was accomplished by abolishing the hearing in the healthy ear only, provided the masker did not have sufficient intensity to substantially affect the hearing of the "worse" ear when the better ear was masked, i. e., as long as the noise transmitted through the head had a smaller intensity than was the apparent hearing loss of the worse ear.

The amount of noise which penetrated into the region of the worse (left) ear was estimated in the following way: The masker was applied against the observer's right ear and the examiner brought his own right ear in contact with the observer's left ear. Over his own left ear the examiner placed the air-conduction receiver and matched certain loudness of the 128 d.v. tone with the intensity of the masking noise as perceived through the observer's head. It was estimated that about 25 to 30 decibels of the instrument's noise penetrates the patient's head. This amount of noise has a sub-threshhold value for the deaf ear under examination which, if the shadow curves 3 and 7 were true curves, would register 70 to 80 sensation units hearing loss. With such a high threshold practically all hearing occurs by bone-conduction no matter whether the air-conduction receiver or the bone-conduction receiver is used for the test.

Second test: (Observer No. 2, Fig. 5.) This observer was 12 years old. Deafness of the right side was first observed when the girl was 10 years old. There was no history of any previous ear symptoms. Past medical history was irrelevant and there was no history of deafness in the family. Otoscopic and nasopharyngoscopic examination revealed no pathology. Both ears had a positive caloric labyrinth test. However, the nystagmic reaction from the right superior canal was delayed. The Gellé test was positive on the left side in the whole range of the audiometric frequencies. The right ear could distinguish rarefaction and compression only if tested with 512 d.v. tone, and this was confirmed by several tests performed on different days. Thus, apart from the hearing test, the caloric reaction indicated that there may be, and the Gellé test that there must be, some cochlear function on the right side.

Audiograms V and VI of Fig. 5 show a moderate decrease of the air-conduction acuity (curves 21 and 22) in the presence of

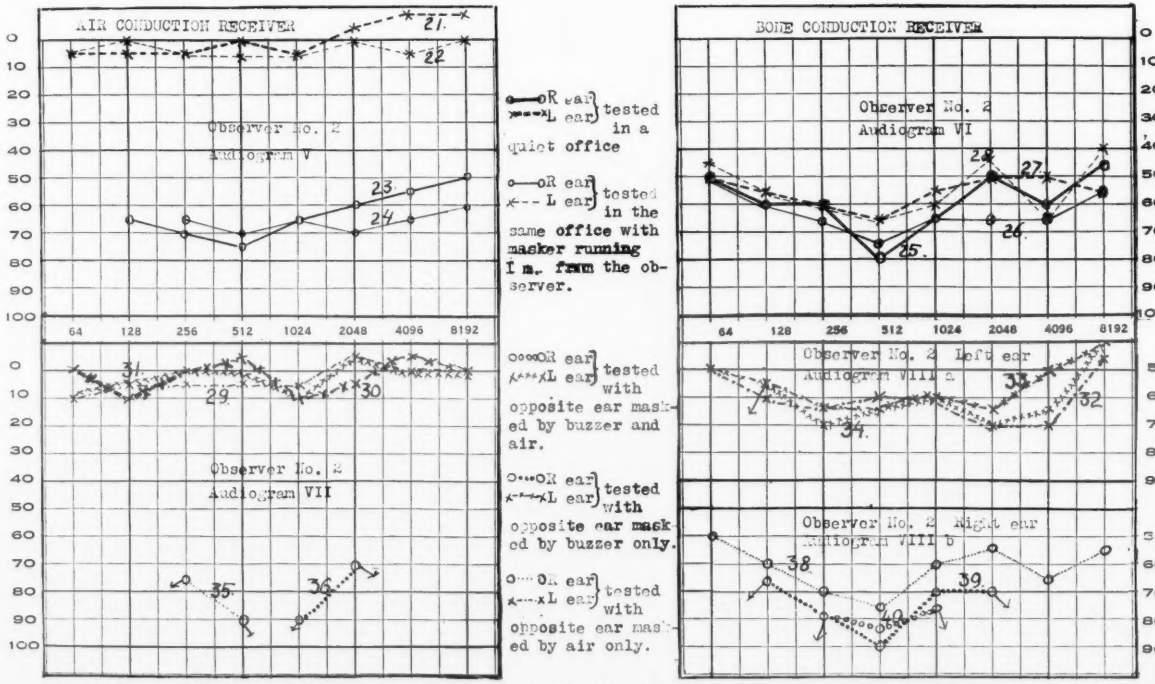

Fig. 5. Test of the masker against observer No. 2, who is affected with deep congenital perception deafness in the right ear.

the extraneous noise of the masker. The effect of the hum on the shadow curves 23 and 25 and on the bone-conduction acuity of the good ear (27) had no definite character. The variations of the noise (curves 24, 26, 28) from the curves taken under better acoustic conditions (23, 25, 28) could best be explained as an "observation disturbed by noise." By such an explanation we designate rather the brain cortex than the peripheral receptor as the location of the disturbance.

Comparison of audiogram V with audiogram VI shows that the bone-conduction shadow curve 25 is almost on the same level as the true bone-conduction curve of the opposite ear 27. This gives an impression that there is a rather extensive function of the right cochlea. On the other hand, while the air was blown into the left ear, practically all perception from the air-conduction receiver disappeared from the right ear (audiogram VII, curve 35). This again gives an impression of no function of the right cochlea. Curve 36 (the masking effect of the buzzer only), gives a similar impression and curve 37 is missing because the combined noise of the masker introduced into the good (left) ear abolished all hearing when the tones of the audiometer were introduced at top intensities into the bad (right) ear through the air-conduction receiver. The shadow curve 23 certainly was not effaced by the 25 or 30 decibels of the masker's noise penetrating from the opposite ear, because, in reversing the experiment, the true curve 21 was altered into curves 29, 30 and 31, with variations indicating only "observation disturbed by noise." In tests 29, 30 and 31 the curve 21 was subjected to disturbance by a noise 30 decibels above its own level. The conditions were quite different in tests 35, 36 and 37. Here the curve 23, if it were a true curve of the hard of hearing ear, was being "disturbed" by a noise 30 decibels below its own level, i. e., by a noise which the defective ear could not hear. Only one explanation remains: Curve 23 is a "shadow-curve," traced on the audiogram while the right ear was being tested, but actually heard in the left ear.

Audiogram VIIIb represents the testing of the deaf (right) ear with a bone-conduction receiver while the good (left) ear was masked. The most striking difference is that between the curves 38 and 35: While the blowing of air into the right ear abolished practically all hearing, in the deaf ear which was listening to the air-conduction receiver (curve 35), the same test with the bone-conduction receiver (curve 38) indicated a striking inefficiency of the streaming air as a method of masking. The masking effect of the buzzer alone (39) was definitely better than that of the air-stream alone. The combined

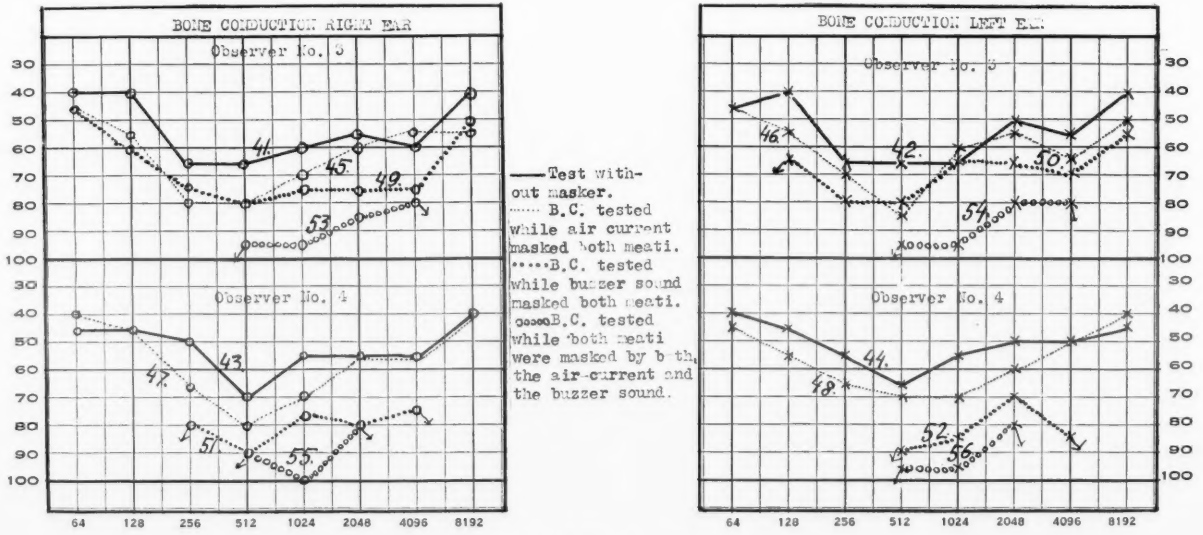

Fig. 6. Test of the masker against two observers No. 3, and No. 4, with normal hearing in both ears. Bone conduction only.

noises (curve 40) allowed hearing in the middle frequencies. In one of these frequencies (512) the Gellé test was positive.

Before we accept the curve 40 as the true bone-conduction curve of the right ear we have to answer one more question: Why this curve or its equivalent did not appear when the air conduction receiver was used in the test? To answer this question we have to make two assumptions: (1) The conduction apparatus of the right ear was insufficient; (2) the air-conduction receiver transmits less energy to the skull tissues than the bone-conduction receiver.

Conclusion of the second test: The observer was selected for testing of the masking apparatus because the diagnosis of deep unilateral deafness with some remnants of the cochlear function could be made without reliance on the audiometer. The graphic records of his hearing indicate that with proper masking methods the shadow-curves can be eliminated and yet traces of cochlear function can be demonstrated in unilateral profound deafness.

How truly such remaining curves depict the pathological changes in the ear and what is the final diagnosis of such cases is not the purpose of this study.

STANDARDIZATION OF THE MASKER

The contact points of the electrical buzzer may need occasional cleaning, the metal spring needs adjustment, the batteries have to be changed, the rubber balloon replaced. After each such adjustment the masker should be checked. For such purpose a record of two easily available observers with normal hearing should be kept on hand. The procedure is represented by Fig. 6 and can be summarized as follows:

1. Record the bone-conduction curve for each ear separately under the ordinary acoustic conditions of the office (curves 41, 42, 43, 44).

2. Introduce the air current into both ears and test again with the bone-conduction receiver (curves 45, 46, 47, 48).

3. In a similar way record the bone-conduction curves when the buzzer only is acting (curves 49, 50, 51, 52).

4. Test the bone conduction of each ear with combined noise introduced into both meatuses (curves 53, 54, 55, 56).

Figure 6 shows that a healthy ear perceives only several top intensities produced by the bone-conduction receiver of the 2B Audio-

meter if the combined noise of the masker is acting on both ear drums.

SUMMARY

The masking apparatus consists of an acoustically insulated electric buzzer combined with a simple air-current device. The instrument produces only a minimum of extraneous noise and about 60 to 65 decibels of effective complex masking tone. Audiograms are presented which describe the elimination of the bone-conduction and the air-conduction shadow curves in one case of total unilateral deafness and in one case of deep unilateral congenital hearing defect. A method is suggested for testing and standardizing of the instrument.

3028 36TH STREET.

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XII

EXPERIMENTS ON THE CONDUCTION OF SOUND IN THE EAR*

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Chicago

It is the purpose of this paper to report on experimental work on sound conduction in the auditory apparatus. The planning and execution of our experiments has been helped to a great extent by the progress in acoustics and other branches of physics. The improvements of the experimental methods enable us today to design and analyze experiments which could not be carried out previously. The development of the electrical sound producer has provided more accurate definition and measurement of sound as well as a reliable method of reproducing the stimulus. Another aid has been the development and perfection of an optical recording system. The vibrations of the ossicles and even the movements of the labyrinthine fluid can be recorded and measured. Both the stimulus and the effect are therefore obtained in true quantitative relationship. The improvement of the method represents a great practical step forward. In addition, important theoretical contributions have been furnished by the developments in modern acoustics. During the last decades inventors have developed the telephone, the microphone, the phonograph and other sound recording devices. Scientists have studied the construction of these instruments and founded the theory of sound recording. The knowledge acquired in the construction of artificial sound recorders can now be used in the analysis of this great natural but mysterious sound receiver which we all carry in our temporal bone.

The analysis of the sound conductive apparatus, which will be given here, differs to a certain extent from former work. The conditions of sound conduction have been generally considered as constant. In this paper an attempt is made to demonstrate that the conditions of sound conduction in the ear are not constant, but they differ according to the quality and quantity of the stimulus. We have come to this conception after having studied a part of the middle ear which has been for a long time neglected.

*Read before the Chicago Laryngological and Otological Society, November 1, 1937.

From the Division of Otolaryngology, the University of Chicago.

The function of the intrinsic muscles of the ear has been given little attention heretofore. It may be remembered that Helmholtz[1] considered the middle ear muscles only as a kind of ligament, an elastic suspension material, incapable of active contractions. Otto Frank,[2] in his work on the ear, which is perhaps the most original study after Helmholtz and Bezold, did not take the muscles into consideration. In the "Handbuch der normalen und pathologischen Physiologie" the muscles of the middle ear are described as "rudimentary organs without any active function."

The first part of this demonstration will show the activity of the muscles in the presence of acoustic stimuli.

The second part will demonstrate the effect of the muscles on sound conduction.

Most of our experiments were carried out in rabbits. In addition, there are observations in the rat, the dog, and also in the human.

In the animal experiment we expose the bulla and open the tympanic space from the ventral side so that destruction of any part of the middle ear is avoidable. The routine position of the rabbit was five inches from the loudspeaker.

Both the frequency and the intensity of the sound are of great importance for the contraction. Low intensities do not produce visible muscle contractions. The threshold of hearing is lower than the threshold of the muscle contraction. The stapedius threshold is slightly lower than the tensor threshold. So far as the frequency is concerned, the middle range frequencies produce the strongest contractions. Lower and higher tones show a higher threshold, producing a curve which resembles the threshold of hearing. Figure 1 shows a tone which produces a definite contraction of both muscles. After a short latency period both muscles rise to a tetanic contraction which equals the duration of the stimulus. The contraction is a true tetanus and not a single jerk as claimed by the first observers. Shortly before the beginning of the tone there is a contraction of the stapedius muscle. This contraction, however, is not the "listening" contraction as postulated by Ostmann.[8] This author proposed as function of the muscle a short contraction immediately before or during the beginning of the acoustic irritation. In reality the "upbeat" of the stapedius muscle as found in these curves is due to a click sound produced by the opening of the camera. The click of the camera happens to be a stimulus above the stapedius threshold, but below the tensor threshold.

In regard to the intensity, three groups of stimuli can be distinguished. First, underthreshold stimuli for both muscles; second, a

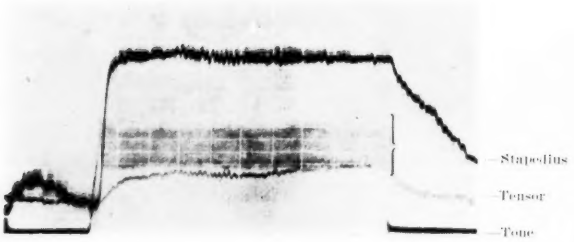

Fig. 1.—Contraction of the tensor tympani muscle and stapedius muscle due to an acoustic stimulus.

narrow range of intensities which are overthreshold for the stapedius; thirdly, overthreshold stimuli for both muscles.

The contraction ends accurately with the determination of the sound. The "dampening theory" cannot be substantiated, i. e., the muscles do not dampen any eventual aftervibrations following an acoustic stimulus. Their effect is definitely simultaneous with, and ends with the tone.

Figure 2 shows an increase of the tone while the recording was done. We see parallel to the "crescendo" of the tone an increase of the muscle contraction.

For many experiments it is easier not to record the contractions, but simply to observe them. The experimentor determines the intensity of a tone which gives a threshold contraction. The readings are taken for different frequencies. Curves are plotted in a similar way to audiometer curves with the difference that the threshold of the contraction is recorded instead of the threshold of hearing sensation.

Figure 3 is a typical normal tensor threshold in a rabbit. The solid line represents the tensor threshold taken under general anesthesia. Our constant procedure consisted in producing shallow anesthesia by intravenous injection of ethyl carbonate. The broken line in this curve is the tensor threshold of an animal in local anesthesia. It can be seen that the difference is negligible.

The reflex contractions can be seen in rabbits, dogs and cats. In the human the observation of the tensor tympani muscle is handicapped by unfavorable anatomical conditions. For this reason no positive observations have been described. The tendon of the sta-

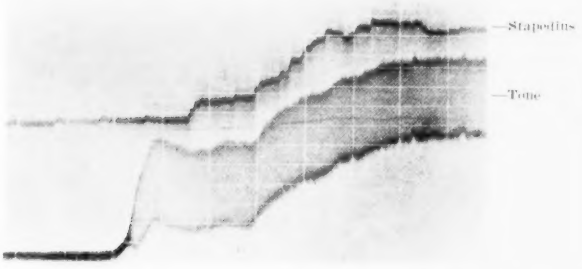

Fig. 2.—The influence of the intensity of the stimulus on the muscle contraction. The stimulus has been increased during the recording. Note the increase of the muscle contraction parallel to the "crescendo" of the tone.

pedius muscle has been successfully observed and an acoustic reflex demonstrated which shows qualities nearly identical with those of the animal. The prerequisite for an observation of the stapedius reflex is a perforation of the ear drum in the posterior-superior quadrant. More than 30 patients have been observed in the University of Chicago Clinics, in whom the stapedius reflex could be demonstrated.

In addition the attempt was made to study the stapedius reflex in the normal ear. With the kind help of the anatomist Prof. Spalteholtz a method was found to make the ear drum transparent in the posterior-superior quadrant. It was designed as a physiological method to determine the qualities of the stapedius reflex in the normal ear. In suitable cases we succeeded in observing the stapedius reflex through such an "optical perforation." Although the percentage of failures is high, this procedure has been used as a physiological method to observe the muscle reflex in the normal ear.

The contractions of the middle ear muscles are simple reflex movements. The N. acusticus is the centripetal part of the reflex which carries the impulse from the cochlea to the primary acoustic nucleus. Destruction of the cochlea or cutting of the eighth nerve abolish the reflex. The central part of the reflex has been located in the pons. The centrifugal branches are the trigeminal nerve for the tensor and the facial nerve for the stapedius.

The reflex is independent from the will.

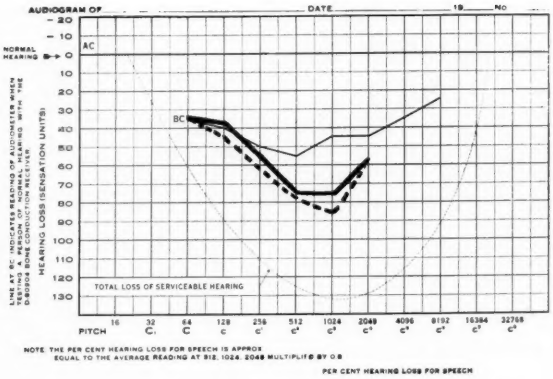

Fig. 3.—Normal tensor threshold in the rabbit; the solid line represents the tensor threshold under shallow general anesthesia. The broken line is the curve taken under local anesthesia. The difference is negligible.

A few human beings are able to contract their intrinsic ear muscles voluntarily. These contractions, however, seem to be of no functional significance.

It is of interest that there is another striated muscle in the human body which works both reflexly and voluntarily. This muscle is the diaphragm. Recent research has revealed that the ear muscles and the diaphragm have several features in common. First, they are all phylogenetically young muscles. Secondly, their pharmacological behavior is different from other muscles. These findings cannot be discussed here, but they may lead to new information on the pathological function of the ear muscles. [4, 5, 6]

The mechanical effect of the muscle contractions has been studied by the following method:

Air pressure changes of sinus form have been produced in the outer acoustic meatus of a fresh human ear specimen. The resultant movements of the malleus are shown by an optical recording system. If the tensor contracts, the vibrations of the malleus are considerably reduced. If one records the movements of the stapes, one can find that the tensor exerts only a slight effect on the stapes. The stapedius muscle works mainly on the stapes, and affects the malleus to a much smaller degree. The mechanical effect of the contractions consists

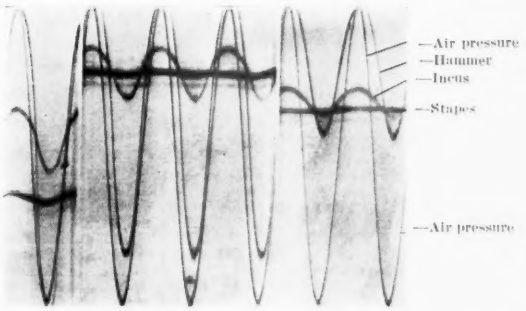

Fig. 4.—The effect of the intrinsic muscles of the ear on the movements of the ossicles. In the experiment rhythmic changes of the air pressure in the outer acoustic canal are produced and recorded. The movements of hammer, incus and stapes are recorded. The left third of the figure shows the movements of the ossicles without muscle contraction. (Underthreshold tone.) The middle part represents a tone which has produced a contraction of the stapedius alone. There is a strong diminution of the stapes amplitude but only a slight effect on the hammer excursions. The third part of the figure demonstrates the effect of a loud tone which produces a contraction of both muscles.

NOTE—The amplitudes of the ossicles diminish from hammer to stapes. In the literature the proportion is given as 4:2:1. In this experiment it is demonstrated that the proportion is not constant. For the underthreshold tone the proportion is 7.7:1.9:0.6. Due to the contraction of the stapedius the proportion is changed to 6.7:1.4:0.1.

in a diminution of the amplitude, the tensor working more on the lateral ossicle, the hammer, the stapedius being effective mainly on the stapes.

Let us now recall that there are three groups of tones (according to their intensity). Soft tones do not produce muscle contractions; a louder tone produces a contraction of the stapedius only, and a still louder tone produces a contraction of both muscles. Thus there are three mechanical conditions in the ossicular chain according to the quantity of the stimulus.

Figure 4 shows these conditions. The sinus wave in the outer ear and the movements of all three ossicles are recorded. The part at the left is the weak, underthreshold tone. Since there is no muscle contraction the movements of the ossicles are undiminished. The

middle part of the recording represents a stimulus which is effective on the stapedius muscle alone. It is evident that the movements of the stapes are diminished to a higher degree than those of the hammer. The effect is a change of the previous proportion of ossicular amplitudes. The third part of this recording demonstrates the movements of the ossicles when both muscles contract. Now the movements of the lateral ossicles are diminished more than those of the stapes.

A second important feature of sound conduction in the ear is the fidelity of recording. In a good microphone good fidelity is secured over a wide range of frequencies. The resonance curve of a good microphone resembles a straight line. In order to determine the resonance curve of the ossicular chain one has to measure the resonance frequency and the dampening factor. The resonance frequency of the ossicular chain was measured after the method of Frank.

The ear specimen is prepared for recording of vibrations of the hammer by attaching a small mirror to the head of the malleus. A sudden change of the air pressure in the outer acoustic meatus is then produced and the resulting vibrations recorded. The ossicular chain is to be considered as an elastic system of given elasticity and mass. Anatomically the elasticity is represented by the ear drum, the ossicular ligaments and the tension produced by the muscles. The mass factor is the sum of the three ossicles. Due to the anatomical arrangement, however, in calculating mass, the lever effect must be taken into consideration.

Figure 5 shows the experimental finding. The horizontal distance between the peaks represents the frequency, the decrease of the amplitudes is used for determining the dampening factor.

The resonance frequency is found between 750 and 800 d.v. The dampening of the ossicular chain, expressed in the "Dampening Factor" of Frank is D = .3.

After having determined the resonance frequency and the dampening one is able to plot the resonance curve. The resonance curve of the middle ear is far from being a straight line. The fidelity is rather poor. Frequencies in the resonance range are over-recorded in the middle ear considerably beyond their true value.

In another series of experiments the influence of the muscle contractions upon the resonance curve was studied. Experiments revealed that the resonance curve of the middle ear remains unchanged by the activity of either the tensor or stapedius muscle.

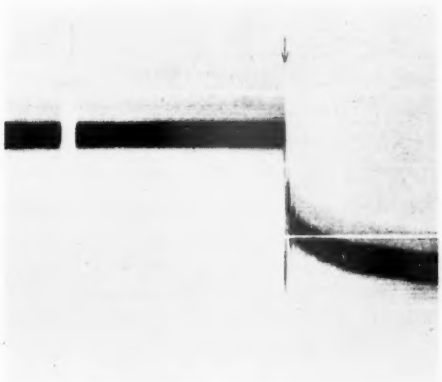

Fig. 5.—Resonance frequency of the ossicular chain. A sudden pressure change in the outer acoustic meatus (indicated by arrow) starts vibrations of the ossicles.

DISCUSSION

These experiments aim to throw light upon two important physiological questions. First, what is the function (or the purpose) of the acoustic reflex of the muscles of the middle ear? Secondly, what is the mechanism by which they execute their function?

Among all experimental observations the diminution of the vibrations of the ossicles, due to the muscular contraction, is the most outstanding finding. In addition, the presence of the reflex contractions during strong stimuli, and their absence in weak tones, point toward a protective function. On the other hand the accommodation theory has found no corroboration, since the resonance frequency of the ossicular chain does not change during the muscular contraction. All the other more or less speculative functions which have been attributed to the muscles, such as "listening" or "dampening" (prevention of after vibrations) or "equalizing of pressure" should be discarded.

However, one should keep in mind that there are nonacoustical contractions of the muscles, either voluntary or involuntary, which should be strictly separated from the acoustic reflex. Unfortunately, great confusion has been created in the literature by uncritical interpretations. The existence of the reflex has for this reason not been

duly recognized, and has even been denied, in spite of excellent observations of various experimenters.

Johannes Mueller, as far back as 100 years ago, had already described the intrinsic muscles as a protective apparatus. However, the evidence remained unconvincing and led to various other theories. The improvements of the experimental method has given a clearer conception of the function of the muscles. The protective theory explains the experimental findings best.

The acoustic stimulus controls the strength of the muscle contraction very accurately. Threshold observations and overthreshold recordings revealed that each acoustic stimulus has a certain constant and regular strength of contraction. The muscle contraction in turn changes the sensitivity of the ossicular chain. Each tone has therefore its own characteristic sensitivity of sound conduction. From these experiments the ear can be described as a sound recording device with changing sensitivity. The conditions of sound conduction are not constant. Each tone travels over an ossicular chain, the sensitivity of which is specially adapted to the physical qualities of the tone.

The variation of the sensitivity is, however, not the only effect of the muscle contractions. It has been stated previously that stimuli close to the resonance frequency are over-recorded. The reflex is found very sensitive in the resonance region. The diminution of the amplitude is there especially effective in this region. The over-recording due to resonance is diminished by the muscle reflex. The muscle reflex acts as a correcting device.

The question may be asked, why did nature build a faulty sound recorder to which a correcting mechanism had to be added? Why is the ear not an instrument with perfect fidelity? The reason is obvious. The sensitivity of the ossicular chain is increased in the resonance zone. Weak stimuli which do not produce muscle contractions utilize the over-recording. They are actually amplified. Underthreshold stimuli may be made audible. The sensitivity of the ear in the most important tone range is therefore enhanced for soft tones. Loud tones which need no amplification are cut down by the muscle reflex.

It is evident that the ear is not built in accordance with the principles of sound recording as developed by science. Artificial sound recorders (microphones, for example) are built with resonance frequency, outside the most important speech frequencies. The middle ear has a resonance frequency located exactly in the area of

the spoken voice. Bezold has called attention to the tone range b_3 to g_4 which today is commonly referred to as Bezold's sixth. The resonance frequency of the middle ear is found within Bezold's sixth.

In our experimental animals the mass of the ossicles is smaller, the resonance frequency is found higher. For the cat, Broemser[7] found the resonance frequency about 1,600. In the rabbit it may be still higher, perhaps 2,000. The principle of construction, however, appears to be the same as in the human: The middle ear is in resonance with the most important acoustic frequencies.

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XIII

THE CLINICAL AVAILABILITY OF THE OSTIUM MAXILLARE: A CLINICAL AND CADAVER STUDY*

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CLEVELAND

In reviewing the literature dealing with the practical clinical availability of the natural ostium of the maxillary sinus, I was surprised to find that more productive and constructive work has been done upon this subject in the last ten years than ever before.

This especially is to be wondered at when we recall that Casserius mentions the maxillary sinus as early as 1612, that Nathaniel Highmore fully described it in 1651, and that Jourdain, a dentist of Bordeaux, in 1765, made the first clinical effort to treat disease of the maxillary sinus by approaching it through its natural opening.

My own particular interest in this matter is of but relatively recent origin inasmuch as my earlier contacts with teachers and colleagues included chiefly those who were satisfied to irrigate the sinus in the usual and accepted fashion by puncture through the inferior meatus. For those who spoke or wrote of successfully irrigating the maxillary sinus through the ostium in 60 to 80 per cent of their sinus patients, I shared with many others a profound skepticism, if not disbelief. It was in this frame of mind that I began to carry an ostium catheter into the anatomical laboratory and to ascertain on the cadaver just how practical this procedure might be in my own hands.

In this connection, I want to quote a paragraph from a paper written by Myerson[1] in 1932: "I was impressed with the fact that none of those who objected to the technique had undertaken to establish by actual clinical application the percentage of maxillary sinuses that one could enter. Most statements for and against appear to be based on inference or conjecture but not on careful clinical trial."

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*Presented at the Middle Section meeting of the American Laryngological, Rhinological and Otological Society at St. Louis, January 26, 1938.

This report is based upon my observations in attempted catheterization of the ostium of the maxillary sinus in 135 cadavers and 70 consecutive private patients suspected of having maxillary sinus disease. For several reasons I could wish that the series of private patients were larger, but only comparatively recently did I become sufficiently interested to state in the patient's record whether the sinus lavage had been done by puncture through the inferior meatus or by catheterizing the ostium.

Through the courtesy of Dr. T. Wingate Todd, Professor of Anatomy at the Medical School of Western Reserve University, I have been studying cadaver heads for several years. Dr. Todd has preserved intact a very large research collection of skeletons, including skulls. During the process of student dissection the skull is disarticulated from the vertebral column and, except for a study of the soft parts, is not further molested by the medical student. Before the head is macerated, the skull is expertly sawed through, always just to the right of the nasal septum near the mid-sagittal plane and its internal aspects are then carefully noted, without, however, in any way altering any of the visible structures such as fracturing or removing turbinates, etc. Although our residents and I have profited for some time from this unusual opportunity of studying the lateral nasal wall, it is only in recent months that I have been attempting to catheterize the maxillary ostium in this fresh cadaver material. At first I endeavored to use the left side of the skull having the attached septum, but this proved unsatisfactory.

The clinical material consisted of seventy consecutive private patients from 13 to 64 years of age who had or were suspected of having maxillary sinus disease. In the seventy patients an attempt was made to irrigate the maxillary sinus in ninety instances, forty-four right antra and forty-six left antra.

In both the cadaver and patient series the Turnbull catheter was used. The distal half of this catheter is made of pliable silver and can be bent into any desired arc, but is still sufficiently rigid to maintain its shape under reasonable stress.

In the cadaver series particular care was exercised in duplicating, in so far as possible, the manual technique used in the patient series. The lateral nasal wall was not first inspected so as actually to visualize the ostium and so prejudice the operator, but on the contrary, the head was held in an upright position and the catheter was introduced parallel and close to the lateral nasal wall and the proximal end of the catheter was not allowed to move medially more than

would have been possible were the nasal septum in its normal position. Another temptation that was studiously avoided was the medial fracture of the middle turbinate. This procedure would have considerably increased the percentage of successful catheterizations, but this, of course, would have nullified the purpose of the study. Both Myerson and Van Alyea[2] have reported their results in catheterizing the maxillary ostium in cadavers, but it is doubtful if either author had such favorable material for study, and neither has reported his exact technique of catheterization.

In the patient series the middle meatus was anesthetized by application of 10 per cent cocain in adrenalin and the actual maneuver of attempted catheterization was carried out as described. An important feature of the Turnbull catheter, and one which facilitates its introduction, is the variation which the operator may make in the angle of the tip as well as the change in the arc of the tip, depending upon the height of the uncinate process and the relative depth of the hiatus as well as the proximity of the adjacent bulla and the tip of the middle turbinate. When necessaray, several changes in the configuration of the instrument will frequently overcome what otherwise would constitute an anatomical obstruction, and will thus contribute to the success of the procedure.

A total of one hundred and thirty-five adult cadaver skulls were examined, of which ninety-four were white and forty-one negro. Because of the previously mentioned method of sectioning the skulls, only the right half of the skull was used for this study. Forty-four (46 per cent) of the white skulls and twenty (48 per cent) of the negro skulls were successfully catheterized, using a rigid clinical technique. This is considerably less than the percentages reported by other investigators. Myerson was successful in 87 per cent of 114 lateral nasal walls and Van Alyea in 81 per cent of 163 specimens. I agree with Van Alyea that the incidence of successful catheterization is apt to be less in the cadaver than in the living, owing to the greater pliability of the tissues in the living. Following the suggestion of Van Alyea of dividing the hiatus into thirds, I found that the ostium occurred in the lower or posterior third in 70 per cent of both the white and negro skulls. Van Alyea's results coincided with these findings. An interesting observation was the comparative incidence of accessory ostia in both negro and white skulls. In the negro an accessory ostium was found twice, or 4 per cent, while in the white it occurred 16 times, or 17 per cent. Various authors give the average incidence of accessory ostia as 10 to 50 per cent.

An attempt was made to catheterize the ostium, either right or left or both, ninety times in seventy private patients. The procedure was successful in 62 instances, or 68 per cent. There was but little difference in the frequency of involvement of either antrum and but slight difference in the success achieved in attempting to catheterize either antrum. It is noteworthy, however, that successful catheterization may be made on one side but fail on the other or even fail on both sides; the former occurred eight times and the latter four times in this series.

Inasmuch as my youngest patient was 13 years of age, I can merely quote the opinion expressed by Goldman[3] and Yankauer, that ostium catheterization is much easier in young children, owing to the relatively larger size of the ostium in children. In 1929 these authors reported successful catheterization in a series of children, the youngest of whom was six weeks of age. Incidentally this anatomical fact of the relatively larger size of the ostium in children may account in part for the lesser incidence of maxillary sinus disease in children as well as for the greater tendency for their spontaneous recovery.

Yankauer[1] emphasized a fact of great practical significance when he pointed out that oftentimes the floor of the orbit extends down to a considerable extent so that its medial convexity comes to lie almost parallel with the outer nasal wall in a position often corresponding to the level of the ostium so that the orbit can easily be injured if a cannula is forced horizontally through the lateral nasal wall in this region. The possibility of this relationship must always be remembered when catheterizing the ostium and especially when doing a middle meatal puncture.

The position of the ostium within the infundibulum is variable, but in the great majority of instances (70 per cent) it lies in the posterior third of the hiatus and its direction may be oblique, horizontal or vertical. The diameter of the ostium may be as little as 1 mm. and as great as 17 by 11 mm. As pointed out by Batson, the ostium may occasionally resemble a canal in those cases in which the distance between the antrum and the lateral nasal wall produces an elongation of the ostium.

An accessory ostium may occasionally be present, and if so usually lies posteriorly, postero-inferiorly or directly inferiorly to the ostium and at distances of 1 to 11 mm. from the ostium and its size approximates the size of the ostium. I have never been aware of having catheterized the accessory ostium in a patient with sinus disease.

COMMENTS

In a discussion of ostium catheterization we must not only consider its feasibility but also, what is even more important to the patient, its desirability. Dintenfass[3] has ably presented this phase of the subject, so that I need only briefly review it.

We may summarize the advantages of ostium catheterization over inferior meatal puncture as follows:

1. Avoidance in a large measure of the various dangers incident to inferior meatal puncture, such as severe hemorrhage and air embolism. I am fully aware that the occurrence of air embolism in this procedure is denied by some investigators, but the sad fact remains that an occasional patient suddenly dies during inferior meatal puncture no matter how careful the surgeon may be. It is admitted also that at times, even after careful ostium catheterization, there is some bleeding, but in none of my patients as yet has it assumed severe proportions.

2. Many claim simplicity as an advantage for inferior meatal puncture. Ostium catheterization is even more simple, for it consists merely in passing an instrument through a hole. I hasten to add, however, that ostium catheterization becomes simple and successful only after the surgeon has taken the time to master thoroughly the intricate anatomy of the lateral nasal wall and has practiced the technique sufficiently (preferably on the cadaver) to acquire a reasonable facility.

3. Avoidance of or at least lessening of shock and pain for the patient. I admit that a careful surgeon can generally perform an inferior meatal puncture with very little pain or shock to his patient. But the same surgeon can accomplish the catheterization, and more often than not the patient will prefer the latter technique, especially if several irrigations are to be done.

Catheterization does have the following disadvantages:

1. It cannot be universally applied. The chief reasons for this deficiency are anatomical. In such cases my own preference is inferior meatal puncture. Because of the possibility of failing to catheterize the ostium at the initial irrigation, I customarily anesthetize both the middle and inferior meatuses of such patients so that no time is lost if one technique proves unsuccessful. Of course, this double anesthetization is done only with patients whom I irrigate for the first time.

2. Lack of certainty that the tip of the cannula is within the antrum. This I have found to be a very practical disadvantage and one which various surgeons have attempted to overcome. Myerson lists the following points as evidence of the proper placement of the cannula:

a. Depth of position of distal end of cannula and its impingement against the ostial borders in movement.

b. Position of proximal end against the septum.

c. Pus return.

d. Type of sound heard when air is introduced through the cannula before or after irrigation.

I have found the following procedure a distinct addition to the aids listed by Myerson. Upon irrigating and noting a clear return flow, I tip the patient's head toward the unirrigated side and inject air through the irrigating system until no more irrigating fluid appears, being careful to keep the cannula in its original position. The patient's head is then tipped toward the irrigated side and is held in such a position as to place the suspected antrum in a dependent position with the ostium uppermost and the anterior naries slightly inferiorly so that the irrigating fluid will drain anteriorly. Irrigating fluid is then again injected from a large record syringe and the appearance of the return flow at the anterior naris is watched for and when observed the total amount thus far injected is noted. Thus the fluid capacity of the sinus is calculated by subtracting the amount required to fill the irrigating system from the total amount used. If the total amount used is greater than the capacity of the system, then it is reasonable to conclude that the difference represents the capacity of the sinus, which constitutes evidence of the proper placement of the cannula. However, if the total amount used is no greater than the capacity of the irrigating system, then it is inferred that the cannula was not properly placed, or, on the contrary, that it may have been within the ostium of the sinus, but that the sinus cavity was pre-empted by some pathological or other process such as degenerated sinus membrane, new growth, cystic formation, etc. In such an event further diagnostic information can be had by iodized oil injection and roentgen examination. The estimation of the sinus capacity is of itself an important diagnostic aid.

3. Various authors cite as additional disadvantages the ill effects likely to follow trauma to the middle meatus and to the ostium. They point out that the search for the ostium may irritate the soft tissues of the middle meatus and lateral nasal wall and thus contribute

to further lack of drainage and ventilation, while any trauma to the margins of the ostium may possibly lead to its narrowing if not total closure. These eventualities exist, but every surgical procedure has its attendant dangers which can be avoided only by adequate training and the exercise of a careful technique. I believe that the search for the ostium should be brief and without the use of any force.

4. The argument that lavage of the sinus through its natural opening is undesirable because of the attendant obstruction to the escape of the sinus contents, especially thick mucopus is a theoretical objection fortunately encountered in practice but seldom.

CONCLUSION

The results of this investigation, together with the findings of others, demonstrates that we are overlooking a technique which is anatomically correct, frequently applicable, not hazardous, informative, and one which certainly is feared less by the patient than puncture.

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XIV

CONTOUR RECONSTRUCTION AFTER EXTERNAL FRONTAL SINUS OPERATION

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SAN FRANCISCO

The magnitude of the surgery, the resulting cicatrices and the amount of bone destruction are the determining factors as to the type and amount of reconstruction necessary to return a frontal defect to its former contour following an external frontal sinus operation.

When the incisions are small, with or without external drainage and a minimum of bone has been removed, excision of the cicatrix with an accurate reposition of tissues using subcuticular silkworm sutures will correct a minor deformity. When the involvement of a frontal sinusitis has been sufficient to require external drainage and there is loss of bony contour from osteomyelitis then the accurate replacement of tissues must be supplemented with a suitable transplant material to correct the deformities of the bony contour.

Cartilage grafts are the material of choice as they are easily obtained, will keep in vitro indefinitely, are pliable, resist infections and lend themselves favorably to sculpturing. I have used auto- iso- and occasionally heterografts (beef) of cartilage for correcting contour defects and all have proved efficient although heterografts should only be used in very exceptional cases because of the uncertainty of their fate. Some absorption of these heterografts will occur; there is more local reaction following their use; they are more susceptible to infection and may even be extruded at times as foreign bodies.

In all large defects of the anterior plate of the frontal bone a one-piece slightly curved cartilage iso- or autograft, beveled at the edges and properly sculptured to reconstruct the supraorbital defect is the most useful transplant to restore the normal contours of the frontal prominence. If the defect is greater than the diameter of the widest unit of cartilage available two or more grafts may be used, but they should be properly beveled, accurately inlaid and implanted as deep under the covering as possible, fitting under the epicranium at the edges of the bony defect. If these precautions are not carried

out the reconstructed area presents a washboard appearance due to the cartilaginous irregularities.

When the contour defect is great and cartilage is required as a filler a heterograft (beef) may be used. This is a preliminary procedure as the graft will be replaced to a certain extent by scar tissue which acts as a filler but loses the accurate contour effect needed. After a six months' waiting period, absorption being at a standstill, cartilage isografts are inserted to bring out the remaining contour irregularities. By this procedure the contour defects are markedly reduced in size and satisfactory readjustments can be made by additional cartilage isografts which would have been insufficient at the first procedure.

No untoward reactions have been noted when hetero- and isografts of cartilage have been superimposed. For this same type of case autogenous dermal grafts with an overlay of cartilage isografts where mass and a firm outer layer are needed have proved very efficacious.

The isografts of cartilage are prepared by removing the perichondrium and preserving the graft in a solution of four parts of normal saline and one part of 1-1000 aqueous merthiolate kept constantly in the refrigerator. These grafts have been used to restore contour defects after remaining so prepared as long as one year. This long waiting period, although not advisable, did not materially affect the cartilage. It was slightly more pliable than when first obtained and microscopically occasionally small vacuoles were noted in the sectioned cartilage. The auto- and heterografts (beef) are preserved in the same manner and the above micro- and macroscopic changes were also noted in these grafts.

For the past five years in reconstructing contour defects of the face and the appendages I have used the cartilage isografts with but very few exceptions. This type of graft is most efficient, highly satisfactory and economical for the patient. It eliminates the pain and disability that accompanies a rib cartilage resection which in itself is often more disabling than the correction of the deformity. The use of cartilage isografts always gives one an abundance of easily obtainable material that is simply sterilized and kept in the refrigerator where it is available at all times. The cartilage isografts can be interchanged regardless of sex, age, color, race or blood grouping. They stand infection well, will not be absorbed and only occasionally cause slightly more local reaction than do autografts. This reaction is not severe and when it subsides the resulting pericartilaginous

Fig. 1

Fig. 2

Fig. 1. Case 1. Bilateral frontal sinusitis with resulting osteomyelitis. Sixteenth day postoperative. Note amount of bony destruction. (Courtesy of Dr. C. C. Bricca.)
Fig. 2. Case 1. Six months' postoperative result following early return of covering flaps. Plastic repair to be done but patient has not returned to date. (Courtesy of Dr. C. C. Bricca.)

fibrosis fixes the graft more firmly in its bed so that no movement is elicited on digital palpation. This is a helpful sequella for it fixes the graft in its proper location. The cartilage isografts bend occasionally but with much less frequency than when using only autografts. This is probably due to the fact that when the perichondrium is removed and the grafts are placed in the merthiolate-saline solution they assume the shapes that the internal strains and stresses of the cartilage cells dictate. Grafts can then be sculptured from this cartilage of the required shape to fit the existing defect. Those few grafts which have bent out of position have probably done so because (1) the graft was too long for the pocket; (2) the external pressure, as measured by the skin elasticity, was not evenly distributed over the graft. If the pressure is greatest on the middle of the graft, one or both ends will be forced upward. The pressure may be greater at the ends of the graft without materially bowing the center as, this being the thickest part of the graft, it yields less readily.

I am presenting two cases of osteomyelitis of the anterior plate of the frontal bone following frontal sinusitis that have come under my observation recently for plastic surgery. The first demonstrates the efficacy of the early return of the covering material and the second shows the contour restoration with cartilage grafts.

REPORT OF CASES

CASE 1.—Dr. C. C. Bricca operated upon this patient, doing a bilateral radical external frontal sinus operation for osteomyelitis of the frontal bones following bilateral frontal sinusitis.

An inverted T incision was made to reflect the forehead covering. Due to the osteomyelitis it was necessary to remove the anterior plate of the frontal bones over both sinuses. When I saw the patient on the 16th postoperative day to consider the problem of plastic surgery, the two skin flaps were turned back and sutured to the scalp. The granulations were healthy, clean and no exposed bone was present. It was decided to pack the wound with aqueous merthiolate for three days and then return the skin flaps to their original position. On the third day Dr. Bricca freshened the skin edges and resutured the skin flaps into their proper location. A midline drain was left at the lower angle of the wound and a large pressure dressing was applied to the forehead. Within ten days the entire wound had healed.

The early return of the large skin flaps markedly reduced the patient's disability and permitted a minimum of postoperative deformity to occur. It had been planned to reconstruct the frontal contour with cartilage isografts in a year's time, but the patient is satisfied with his original result and to date has not returned for reconstruction. The early return of the covering material cannot be accomplished unless the infection is controlled, there is no exposed bone, the granulations are healthy and the existing pockets can be eliminated in the approximation of the covering flaps.

Fig. 3

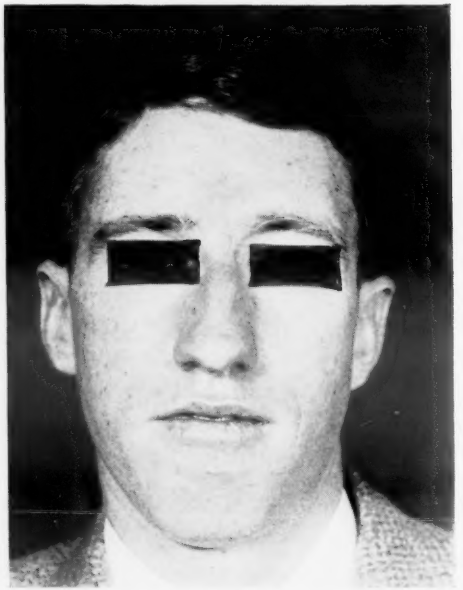

Fig. 4

Fig. 3. Case 2. Unilateral frontal sinusitis with osteomyelitis, eight weeks' postoperative, referred by Dr. R. Fletcher. Notice opening into right frontal sinus.

Fig. 4. Case 2. Postoperative result following return of covering flap and bony contour reconstruction with refrigerated cartilage isograft.

CASE 2.—A patient was referred by Dr. R. Fletcher on whom he had done a unilateral external frontal sinus operation for frontal sinusitis with osteomyelitis. It was necessary for him to remove the anterior plate of the frontal bone over the left frontal sinus. I saw the patient eight weeks after the original operation to consider the problem of plastic closure. Earlier closure was not attempted due to the exposed infected bone, an open right frontal sinus and persistent virulent infection of the soft tissue. At the time of my examination the infection had subsided and was chronic; the bone was covered with granulations which were healthy, the right frontal sinus was still open and the reflected skin was markedly contracted towards the hair line.

The wound was packed with aqueous merthiolate for three days and then under local anesthesia a flap of granulation tissue on a pedicle was turned in to plug the external opening of the right frontal sinus, the edges of the opening having previously been freshened. The skin flap was then dissected free and sutured into its original bed. A small drain was placed at the most dependent angle and a pressure dressing applied. Union occurred throughout the whole flap except at the tip where a small sinus remained. In two weeks time on probing a blind pouch was found that did not connect with the right frontal sinus, so under local anesthesia the surrounding skin was elevated, a small pedicle of granulation tissue was turned in to fill the pocket and the skin closed tight. Primary union of this wound occurred.

Due to the infection, contour reconstruction was not done until eight months after the wound had completely healed.

To correct the deformity an impression of the defect was taken in dental modeling compound (Stent) and then a suitable piece of cartilage isograft prepared as described above was sculptured to correspond to the model of the defect. Under local anesthesia the midline scar was removed and the skin covering the bony defect was elevated. Into this pocket was placed the prepared cartilage graft, the skin was closed and a pressure dressing applied. Some sanguineous drainage occurred at the lower end of the wound due to a collection of blood in the space between the graft and the posterior bony wall. This lasted, however, only fourteen days, when complete healing occurred.

COMMENTS

(1) The early return of soft tissue flaps should be done where possible, for this will afford a good covering, decrease disability and minimize the cosmetic deformity.

(2) Iso- and autografts of cartilage are ideal for contour repair. Heterografts may be used, but only as the exception, and they are not very reliable.

(3) The external closure for an exposed frontal sinus is recorded with no untoward effects and no return of symptoms to date.

490 POST STREET.

XV

OTOMYCOSIS: SOME REMARKS CONCERNING ITS PREVALENCE, SYMPTOMATOLOGY AND TREATMENT*

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SAN ANTONIO

Infection of the skin of the external auditory canal or the pinna with various pathogenic fungi gives rise to a condition which has been designated as otomycosis. It constitutes a minor aural disease, and is the type of mycotic infection most frequently encountered in otological practice. The itching ear, in which the canal is filled with exfoliated epithelial debris of caseous or semicaseous consistency, is a familiar picture to most otologists, regardless of where they are practicing. Heat and moisture have an appreciable effect on the resistance of the skin to invasion, and the degree of pathological change produced by fungi growing within the external auditory canal is greater where climate creates the optimum cultural conditions, but it must be borne in mind that the various fungi producing mycotic otitis externa are capable of growing under adverse conditions with varying degrees of luxuriance. The condition is widely distributed and is more prevalent in the warm, moist climate of tropical and subtropical zones, but it is a mistake to believe that it is confined exclusively to such areas. The condition is apparently not as prevalent in colder climates where the temperature acts as a restraining influence to the growth of fungi.

The usual organisms causing otomycosis are the aspergilli, penicillia, the pathogenic yeasts, the mucoracea and certain of the tinea group of organisms. The condition is easily recognized, but if the changes in the external auditory canal are slight or if the infection is of short duration, the diagnosis may be missed. Examination of smears from the affected ear will often give a clue to the nature of the disease, but exact classification of the infecting fungus is possible only by cultural studies. Cultures made from areas suspected of being mycotic in nature should not be discarded or reported negative until a two weeks' period has elapsed, for many of the fungi do not grow rapidly and may be missed entirely unless this point is

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observed. Saboraud's medium is satisfactory for most fungi, but there are others available which may be used with equally satisfactory results. For the growth of fungi, the medium used must be of high carbohydrate content and rather highly acid. We have found slide cultures of great service in studying the filamentous fungi. Ordinary plate cultures may be utilized in the absence of material for the preparation of slide cultures, but from such cultures, identification is more difficult and time consuming, and if photomicrographs are desired, slide cultures must be resorted to.

The symptoms of otomycosis do not warrant an extended description. Suffice it to say that the sensation of a foreign body in the auditory canal with associated itching, at times intense, are the usual findings. If the epithelial mass in the canal is sufficiently large, hearing may be interfered with. Perforation of the drum membrane attributable to the action of the fungi has not been observed in my practice, and though it has been recorded, it must be of rare occurrence. I have seen myringotomy performed in mycotic infections of the auditory canal due to misinterpretation of the redness of the drum membrane caused by the mycotic process when the debris associated with it has been cleared away. This mistake should be easily avoided. Pain is a prominent symptom when deep invasion of the skin of the canal occurs and with the advent of secondary pyogenic infection, it is intensified and may become agonizing in its severity.

The presence of cerumen in the ear apparently exerts a restraining influence to invasion with fungi, but this is not always true, for instances have been encountered in which fungi were actually growing on a ceruminous plug, though its appearance was somewhat altered. I have encountered mycotic infections in conjunction with chronic otitis media, though profuse purulent discharge from an ear masks the presence of associated mycotic infection.

The sequence of pathological events in otomycosis is somewhat in the following order: Implantation of the organism on the epithelium of the external auditory canal occurs which is followed by a dormant stage which varies from a few days up to two weeks. Then the stage of proliferation comes on, and the fungus grows actively with associated irritation of the superficial layers of epithelium in the canal. If it progresses further, it becomes an actual inflammation in which the top layers of epithelium are exfoliated and mix with the mycotic growth to produce the mass of debris characteristic of otomycosis. It is in this stage of the pathological process that itching is a prominent symptom. It is attributed to the action of the

infecting fungi and the toxins which are elaborated in their growth. It has been suggested that hypersensitiveness to the elaborated toxins may be responsible in some degree for the intense itching and marked changes sometimes encountered. If the infection is not brought under control, penetration of the subepithelial structures takes place, usually in conjunction with secondary pyogenic invasion which gives rise to suppuration manifesting itself as a pyogenic dermatitis, furunculosis, or perichondritis. The cartilaginous portion of the ear may become involved and marked destruction follow. In the suppurative stages pain is present which is often sufficiently intense to require narcotics for its relief. Ulceration of the infected areas may occur where the integument is subjected to the action of macerating secretions. Long standing fungus infections of the ear produce thickening or lichenification of the skin and eczematoid changes may supervene. Extension onto skin surfaces of the face and neck often takes place and may become widespread. Such involvement results from improper treatment, neglect or failure to recognize the true nature of the condition from which the patient is suffering, although the patient's degree of immunity, as well as the virulence of the infecting organism, must be considered in such instances. Cicatricial changes follow in the wake of the more severe mycotic infections of the ear and the subsequent contraction of cicatricial tissue may give rise to discomfort in the involved area which is most rebellious to treatment. The patient with otomycosis does not necessarily run the entire gamut of pathological events noted above, as proper treatment will arrest the progress of the disease at any point in its course. Involvement of the external ear and the postauricular sulcus provides a type of mycotic dermatitis which is at times most difficult to eradicate.

Conditions which may be confused with otomycosis and which must be considered in the differential diagnosis are seborrheic dermatitis, eczematous conditions affecting the ear, tinea infections, particularly associated with tinea infections of the feet; impetigo contagiosa, furunculosis, malignancies, dermatitis venenata, chemical dermatitis and local drug allergies. Foreign bodies may give rise to irritation and swelling of the canal which may be puzzling at times. Plugs of cotton which the patient may have inserted into the external auditory canal and forgotten may set up an inflammatory reaction and produce a certain amount of discharge through mechanical irritation and cause a condition which may be momentarily confusing.

Otomycosis, while not dangerous to the patient's life, is a source of much annoyance and discomfort. Reinfection is frequent, and in certain regions it is almost axiomatic that the reopening of swimming

pools is a harbinger of the mycotic ear infections of the warmer months.

TREATMENT

An early and exact diagnosis of mycotic disease will enable one to apply unrelentingly such measures as we have at our disposal to bring them under control.

Vaccines or filtrates from cultures of the responsible organisms have been used with varying degrees of success. My personal experience with vaccines in mycotic infections has been limited, but the impression has been gained that they possess a definite degree of effectiveness and should be employed as an adjunct to other forms of therapy, particularly where the infection has proved resistant to the usual forms of treatment.

In the therapy of otomycosis, the measures employed are relatively simple, and, if properly carried out, are productive of results which are entirely satisfactory. In the past a great variety of fungicides and fungistatic preparations have been employed, some of which exhibit a marked degree of effectiveness and others practically none. McBurney and Searcy[1] in a recent article have reported both the fungicidal and bactericidal properties of a great many substances used in treating otomycosis, and have pointed out that a large number of preparations are in common use which have little or no therapeutic value so far as this disease is concerned. Their conclusions were based on experimental, as well as clinical investigations. King Gill[2] was the first to call attention to the high degree of clinical effectiveness of metacresylacetate in otomycosis. This was substantiated by the experimental work of McBurney and Searcy, who verified the opinion that metacresylacetate or cresatin heads the list in degree of effectiveness as a fungicide in otomycosis, and further observed that its therapeutic value is slightly enhanced by the addition of thymol. In our experience, the drugs which have been most useful, in the order of their clinical effectiveness, are as follows: Metacresylacetate or cresatin, tricresol 2 per cent in glycerin, Castellani's solution,* iodine in liquid petrolatum or boric acid powder, phenyl mercuric nitrate 1/3000 alcoholic solution, copper salts, such as copper sulphate, chloride, and oleate. Thymol ranks high in fungicidal power being about as effective as tricresol and almost as much so as metacresylacetate, but many patients object to the burning sen-

*Castellani's solution is a saturated alcoholic solution of basic fuchsin to which 10 per cent resorcinal has been added. It also contains phenol, boric acid and acetone.

sation which follows its application. This may be mitigated by admixture of oil, powder bases, or by varying the concentration of thymol. McBurney and Searcy advise the combination of thymol with metacresylacetate. A fungicidal powder* which is a combination of tricresol 1 per cent, iodine 2 per cent and thymol 1 per cent in boric acid powder has proved useful in our hands in the treatment of otomycosis when the use of a powder by insufflation is desirable. It does not possess the sedative properties of metacresylacetate, but is particularly indicated where an absorbent, long-acting fungicide is desired. We have used a stronger powder than this, but we find that most patients object to the temporary irritation which it causes.

The essential points in the treatment of otomycosis are: First, the control of pain; second, the control of the mycotic infection; and third, the prevention of reinfection. Where secondary pyogenic infection is present it must also receive attention. Quite often the external auditory canal cannot be adequately cleansed on the patient's first visit to the physician, because of the pain which manipulation causes. It is in such instances that metacresylacetate serves a two-fold purpose, as a local analgesic and as a fungicide. A cotton wick saturated with this drug can be inserted into the depths of the canal and kept moist by reapplication of the drug at intervals. It is usually advisable to add a vegetable oil, such as olive oil, to the prescription which the patient is to use in this manner, as pure metacresylacetate may cause undue exfoliation of surface epithelium. The wick is removed from the external auditory canal in twenty-four hours, at which time the surface epithelium will be white in appearance and loosened, so that it may be easily removed by swabbing. Knowles[3] has found gentle blowing of compressed air through a small bent metal tube to be of service in the cleansing process.

Occasionally recourse may be had to irrigation with water, but the introduction of water into the auditory canal in mycotic infections, unless it is carefully removed after it has served its purpose,

*Fungicidal powder.

Formula: Iodine, 2.00 grams; Tricresol, 1.00 gram; Thymol, 1.00 gram; Boric acid powder, 100.00 grams.

Mix the iodine, tricresol and thymol together in 30 cc. of 95 per cent alcohol. Spread boric acid powder on a glass slab and add the active ingredients drop by drop to the powder, triturating same to thoroughly mix the active ingredients and the powder and allow the alcohol to evaporate. When the powder is dry, store in dark, glass stoppered bottles. There is some diminution in strength with the passing of time. We have employed several formulas with higher percentages of thymol and tricresol, but we use the above strength by preference because of its less irritant properties.

will stimulate the growth of fungi and often causes an increase in symptoms.

After the second or third application of metacresylacetate on a cotton wick at intervals of twenty-four hours, the canal can usually be adequately and painlessly cleansed, and it is then advisable to change to some other preparation, such as iodine 1 per cent or 2 per cent in liquid petrolatum, or some of the less irritating fungicides noted above. When exudative eczematous changes are present in addition to the mycotic dermatitis, they will require additional treatment which is carried out along the usual well recognized lines. The internal administration of calcium in conjunction with parathyroid gland substance and the use of some type of reducing agent, such as oil of cade in ointment form or mixed with a vegetable oil, will be of service in such instances.

When pyogenic involvement is present as a complication, attention must be given to this through additional measures such as vaccines, local heat, staphylococcus bacteriophage when adaptable, and sedatives for the control of pain. Incision into infected tissue is not often required, and when contemplated should be undertaken with caution, as it may facilitate extension into the cartilage of the ear and may give rise to troublesome consequences. When the bacillus pyocyaneus is present as a co-infecting organism, 2 per cent aqueous solution of acetic acid is recommended, as it renders the epithelium unfavorable for the growth of this organism.

We have used many of the medicinal dyes in the treatment of otomycosis, a few of them being gentian violet, bismuth violet, methylene blue, malachite green and acriflavine. We do not believe that they measure up to other substances in their fungicidal properties.

Prevention of reinfection is important, and if swimming is indulged in, the ears should be carefully dried afterward and 70 per cent ethyl alcohol instilled into them as a prophylactic measure. Weak cyanide of mercury (1:5000) in alcohol is of greater value in this respect, but caution must be exercised in its use and distribution. A safeguard which will often prevent the use of such alcoholic solutions for beverage purposes is the addition of coloring matter, such as methylene blue, to the solution.

Where radiant energy, such as x-ray and radium are employed in treatment of otomycosis, one should not overlook the suppressing effect which these agents have on the ceruminous glands in the skin of the external auditory canal. Ultraviolet light has occasionally been

of service in my hands to alleviate itching. X-ray is indicated in resistant cases, both for its sedative effect on itching and also for the reason that it exerts a restraining effect on sporulation, which in turn influences the life cycle of most of the fungi found as infecting organisms in otitis externa of a mycotic nature.

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XVI

COMPARATIVE ANATOMY OF THE TONGUE*

GREGOR MCGREGOR, M.D.

TORONTO

It seems strange that the tongue is the organ most frequently seen by the laryngologist and is most neglected. It is usually covered up by the tongue depressor because it is a nuisance and in the way. Laryngologists work from above down and from below up to the tongue, but so often when there is real trouble there, the patient is referred to the dermatologist or the general surgeon.

Anatomically the tongue is found to be one of the most variable organs of the body, both in structure and in function, as its development is studied through the various orders of animals from the most primitive to the most completely developed. It varies all the way from merely a fleshy fold in the floor of the mouth, incapable of motion, to a highly developed prehensile organ possessing great extensibility, flexibility, and extraordinary movement. This variation is in keeping with the general law that the more frequent employment of an organ, or part, leads to a proportional increase of development of that part.

The subject matter will be discussed under the following headings:

1. The adult human tongue.
2. The development of the tongue.
3. The hyoid apparatus.
4. Variations in form and movement.
5. The tongue as an organ of taste.

The Adult Human Tongue.—It will be helpful at this point to briefly recall the structure of the human tongue (Fig. 9). It is a median muscular organ attached to the floor of the mouth, the symphysis of the lower jaw and the body and both horns of the hyoid bone, and is covered with mucous membrane. Mucous and serous glands lie embedded beneath the surface.

*Presented before the fifty-ninth annual congress of the American Laryngological Association, Atlantic City, May 31, 1937.

The framework is the hyoid bone (Fig. 24), which is a U-shaped bone consisting of a central body and a pair of greater and lesser horns which serve as attachment for the various muscles which control to a large extent the movements of the tongue.

A large proportion of the tongue consists of the extrinsic muscles (Fig. 10), which pass from the skull or hyoid bone to the tongue. The genioglossus depresses and protrudes the tongue, the hyoglossus depresses it, and the styloglossus draws it backwards or laterally. Associated with these are the elevators of the hyoid bone namely, the stylohoid, the mylohyoid, and the geniohyoid. These are in pairs and symmetrical. The intrinsic muscles, the lingualis, transversus, and perpendicularis, constitute the remainder of the bulk of the tongue, and are responsible for its more delicate and finer movements.

The mucous membrane of the dorsum of the tongue is divisible into two distinct regions, the anterior two-thirds and the posterior third. The line of demarcation is recognized by the V-shaped arrangement of the circumvallate papillæ, at the apex of which is the foramen caecum. The surface is closely beset with papillæ elevations of three varieties (Fig. 25), the filiform, fungiform, and circumvallate. Each consists of a central core of connective tissue stroma covered with stratified squamous epithelium. The anterior two-thirds is rough with fungiform and filiform papillæ. The former, less numerous, appear as red points chiefly near the edges while filiform are everywhere, but arranged more or less in parallel rows. Each circumvallate papilla is surrounded by a depression, and they usually number nine or ten. The taste-buds lie embedded within the epithelium lining the groove encircling the central elevation of each papilla. The posterior third is smooth and studded with masses of lymphoid tissue sometimes called the lingual tonsil.

Development of the Tongue.—Two parts are to be recognized in the tongue (Fig. 11), the buccal part and the pharyngeal part. The buccal part is situated in front of the foramen-caecum and the V-shaped groove, the apex of which marks the site from which the thyroid outgrowth takes place. This part is covered by papillæ concerned in mastication and is susceptible to cancer, as it has the same origin as the lower lip. The posterior or pharyngeal part bounding the buccal wall of the pharynx is covered by glandular and lymphoid tissue, and is concerned with swallowing. These two parts are not only different in function but are different in origin and development.

The buccal part arises during the fourth week by an upgrowth—the tuberculum impar—which springs from the floor of the pharynx in front of the united ventral ends of the second and third arches

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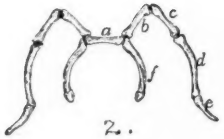

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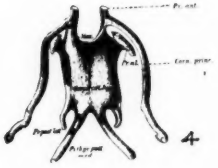
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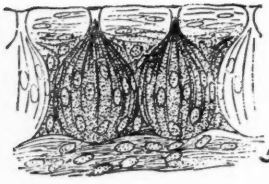
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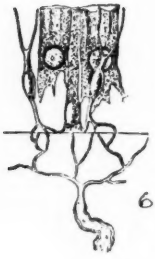
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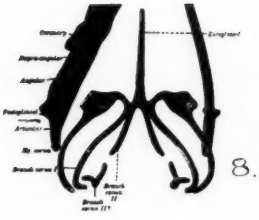
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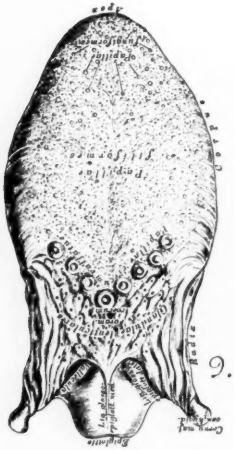
(Fig. 26) and by right and left lingual buds from the mandibular or first arch. Hence the buccal part is partially bilateral in origin. In the seventh week the tip of the tongue is bifid because these lateral buds are imperfectly fused. This bilateral origin of the buccal part explains the occasional occurrence of a bifid tip and the formation of cysts in the median raphé. This part is supplied by the lingual nerve and the chorda-tympani branch of the facial, which enters the mandibular arch. The pharyngeal part is derived from the fused ventral ends of the second and third arches in which the body of the hyoid bone is developed. The glossopharyngeal or nerve of the third arch supplies it. The V-shaped groove marks the union of the buccal with the pharyngeal part. Thus the tongue embryologically has two distinct origins each with its own nerve supply.

The Hyoid Apparatus.—The hyoid bone proper develops from the ventral portion of the hyoid or second visceral arch. In general it consists of a median body or basihyal, representing the fused ventral parts of the second and third arches, two lesser horns or ceratohyals, which may consist of several parts and which serve to unite the hyoid with the base of the skull, and two greater horns or thryohyals, which join the hyoid to the thyroid cartilage. In some species another horn called a glossohyal extends forward in the midline into the base of the tongue, acting as an additional support and in others a horn extends backward and is known as the urohyal.

The lesser horns, or ceratohyals, consist of several segments (Fig. 27). The tympanohyal is embryonically continuous with the ear ossicles and becomes embedded in the petromastoid. The stylohyal ossifies in the early years of life and becomes joined to the tympanohyal to form the styloid process. The segment below, the epihyal, becomes ligamentous and forms the stylohyoid ligament but it may also become ossified. The lowest segment, the ceratohyal, forms the small horn of the hyoid. The epihyal lies behind and outside the tonsil, and when ossified has been excised under the belief that it was some foreign structure. A ridge of bone on the upper surface of the body of the hyoid lying between the lesser horns appears to represent the body of the second arch. It may be separated from the body of the hyoid by a foramen for the passage of a remnant of the thyroglossal duct.

This hyobranchial support of the tongue is seen to undergo some remarkable modifications when traced through the various groups of vertebrate life. In the fishes (Fig. 12) it is a large compressed lamelliform bone composed of a stylohyal, or epihyal, a ceratohyal and a thyrohyal to which may be articulated a glossohyal and a urohyal.



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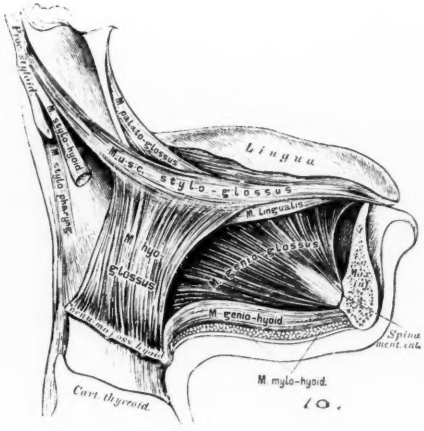
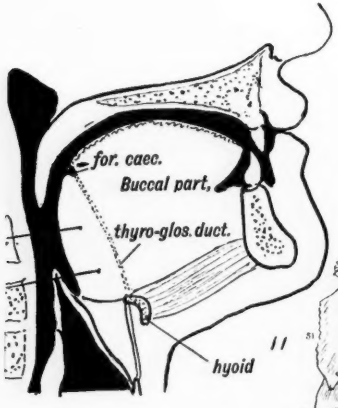

Lingua
M. Lingualis
M. hyo-glossus
M. genio-hyoid
M. mylo-hyoid.
Cart. thyreoid.

10.


for. caec.
Buccal part,
thyro-glos. duct.
hyoid

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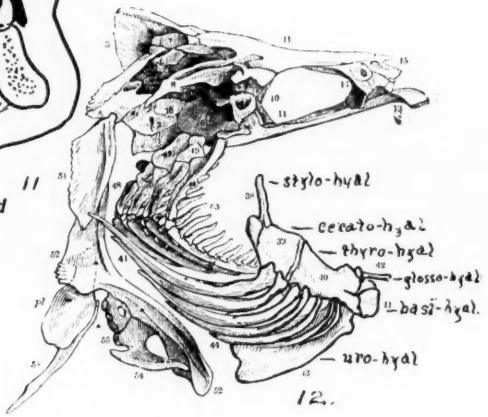

stylo-hyal
cerato-hyal
thyro-hyal
glosso-hyal
basi-hyal.
uro-hyal

12.

Seven branchiostegal rays are articulated to the epihyals and ceratohyals.

Although the fishes possess no functional tongue the material out of which a tongue is to be constructed is present in the form of the anterior part of the hyobranchial apparatus.

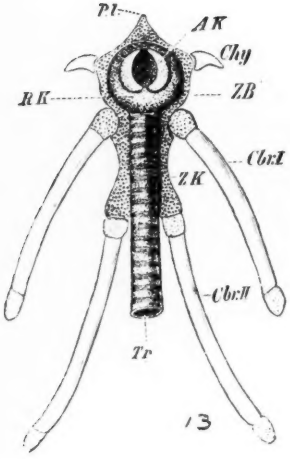
When in amphibians (Fig. 4) these parts become relieved from the gill bearing function, they form the basis of a tongue consisting of a median basibranchial piece, two pairs of posteriorly projecting horns and various processes. When the principal motion is a protrusion and withdrawal of the organ as a whole it is effected by means of the two posterior cornua or horns which lie in sheaths from which they may be everted. When the tongue is unusually long the enclosed cornua are of corresponding length, and may even, as in Spelerpes fuscus, run down the sides of the body and be attached to the ilia.

In reptiles (Fig. 8) the hyobranchial skeleton consists of a narrow basal portion extending forward into the tongue from which three pairs of horns may arise belonging respectively to the hyoid and branchial arches two and three (Fig. 13, water tortoise).

In birds (Fig. 14) the hyobranchial skeleton is greatly reduced. The median body consists of an entoglossal (basihyal) passing anteriorly into a primarily paired paraglossal which extends into the tongue and posteriorly into a urohyal. The single pair of cornua belong to the first branchial arch and may give rise to long pointed rods extending over the skull or as in the woodpecker (Fig. 28) curving around the occipital region passing over the top of the head and terminating at the base of the upper beak near the nares.

The hyoid arch in mammals (Fig. 29) varies greatly. It is connected above by the stylohyal with the base of the auditory capsule and below with the first branchial arch. The part connected with the otic capsule forms the styloid process, while the ventral end becomes the lesser horn of the hyoid bone. The portion between the styloid process and the lesser horn is usually reduced to a fibrous band, the stylohyoid ligament, or it may ossify as epihyal, ceratohyal and hypohyal. Thus the anterior horns, though longer and more complex than the others are spoken of in man as the lesser horns.

In the horse (Fig. 15) the stylohyals, long and rib-like, articulate by a rounded head to the petromastoid: they articulate by means of a short epihyal with the ceratohyals which unite with the basihyal where this is joined to the thyrohyals. The basihyal supports a fairly long glossohyal but in the sheep it is practically absent. The ox (Fig. 3) has a much shorter glossohyal than the horse. In the lion a long liga-



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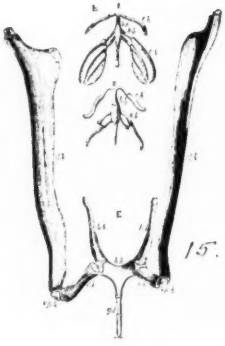
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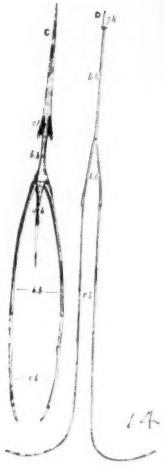
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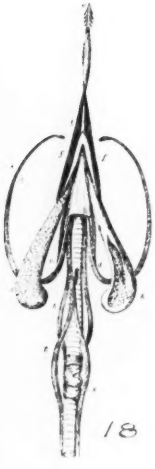
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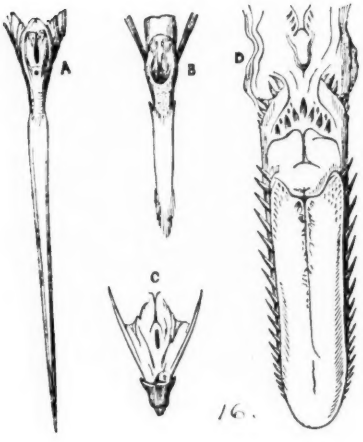


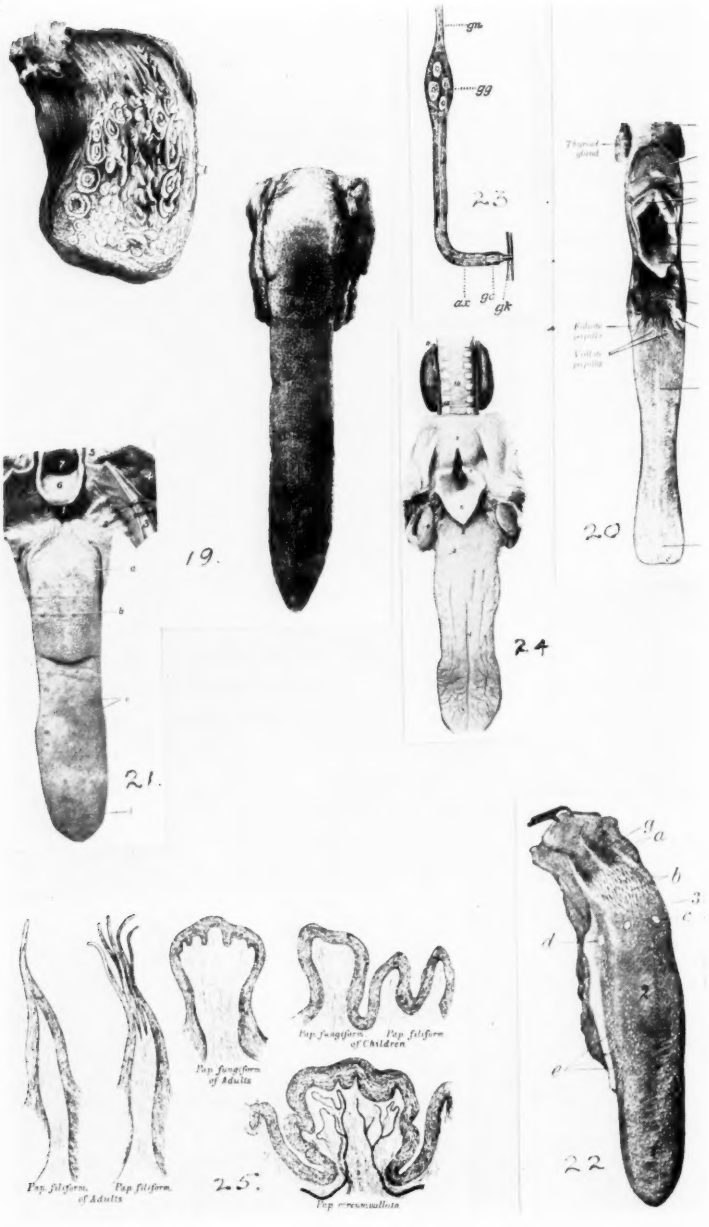
16.

ment intervenes between the stylohyal and the ceratohyals allowing more freedom of motion to the base of the tongue and larynx in relation to the characteristic roar of the King of Beasts.

Variations in Form and Movement.—The tongue is rudimentary in fishes and as a rule is simply represented by a fold of mucous membrane covering the basihyoid, which in all higher vertebrates serves as a point of origin for many of the lingual muscles. It is incapable of motion, except as moved by the supporting skeleton, for it lacks intrinsic muscles. It is sensory, having both tactile and gustatory functions and in some it is provided with teeth. In the lamprey (Fig. 30) it can be protruded and retracted like a piston when the sucker is attached to the prey and it is armed by small serrate teeth for tearing the flesh. It is innervated by the hypoglossal nerve.

In amphibians there is a greater range of structure. In a few the tongue is practically absent and in some it is scarcely more advanced than the fishes but in others it contains intrinsic muscles innervated by the hypoglossal nerve and is extremely mobile. It is attached at the margin of the symphysis of the jaw, its free end, when at rest, being folded back on the floor of the mouth. (Fig. 31.) It can be thrown out with remarkable rapidity with a hinge-like action faster than the eye can follow and for as far as two-thirds of the length of the animal's body. The genioglossi, having their fixed points at the symphysis, raise and jerk forward the free part of the tongue. The return movements are due to the hypoglossi acting from the hyoid arch. The tongue is covered with a slimy sticky secretion which is very useful in capturing its prey.

The reptilian tongue includes not only the parts found in the amphibia but also a median growth, the tuberculum impar, arising between the basihyal and the lower jaw and also a pair of lateral folds lying above the first arch. With the development of these parts the trigeminal nerve also supplies the tongue in addition to the hypoglossal and glossopharyngeal of the lower groups. In turtles and crocodiles the tongue lies on the floor of the mouth and is not protrusible. In snakes and lizards it can be extended from the mouth and withdrawn into a sheath. It is very long, slender, cylindrical and protractible, consisting of a pair of muscular cylinders in close connection along the basal two-thirds, but separated from each other and tapering each to a point at the anterior third. These are in constant vibration when the tongue is protruded. The ununited symphysis of the jaw leaves a passage for the tongue without the need of opening the mouth. The tongue takes no part in the prehension of food other than assisting in the act of drinking.


gn
gg
23
ax
gc
gk
Thyroid gland
Fillate papillæ
Villi de papillæ
20
19.
24
21.
Pap. filiformis of Adults
Pap. filiformis of Adults
25.
Pap. fungiform of Adults
Pap. fungiform of Children
Pap. filiformis
Pap. circumvallata
22

The arboreal chameleons (Fig. 33), clinging on all fours to their tree, depend wholly on their extensile tongue for the prehension of their insect food.

The back part of the tongue of the crocodile (Fig. 34) can be raised against the palate, thus closing the back part of the mouth, so that when it holds submerged a drowning prey the water traversing the mouth has no access to the glottis.

In lizards the shape of the tongue varies so greatly that the differences are used as a basis of classification.

In birds the tongue has lost the lateral parts of the reptilian tongue and with this the trigeminal nerve. It contains no intrinsic muscles. It varies greatly in form, but usually it is slender and is covered with horny papillæ and can scarcely be considered an organ of taste as it is destitute of gustatory papillæ, except at the base, where it is served by a few filaments of the glosso-pharyngeal nerve. It is principally adapted to fulfill the offices of a prehensile organ in association with the beak.

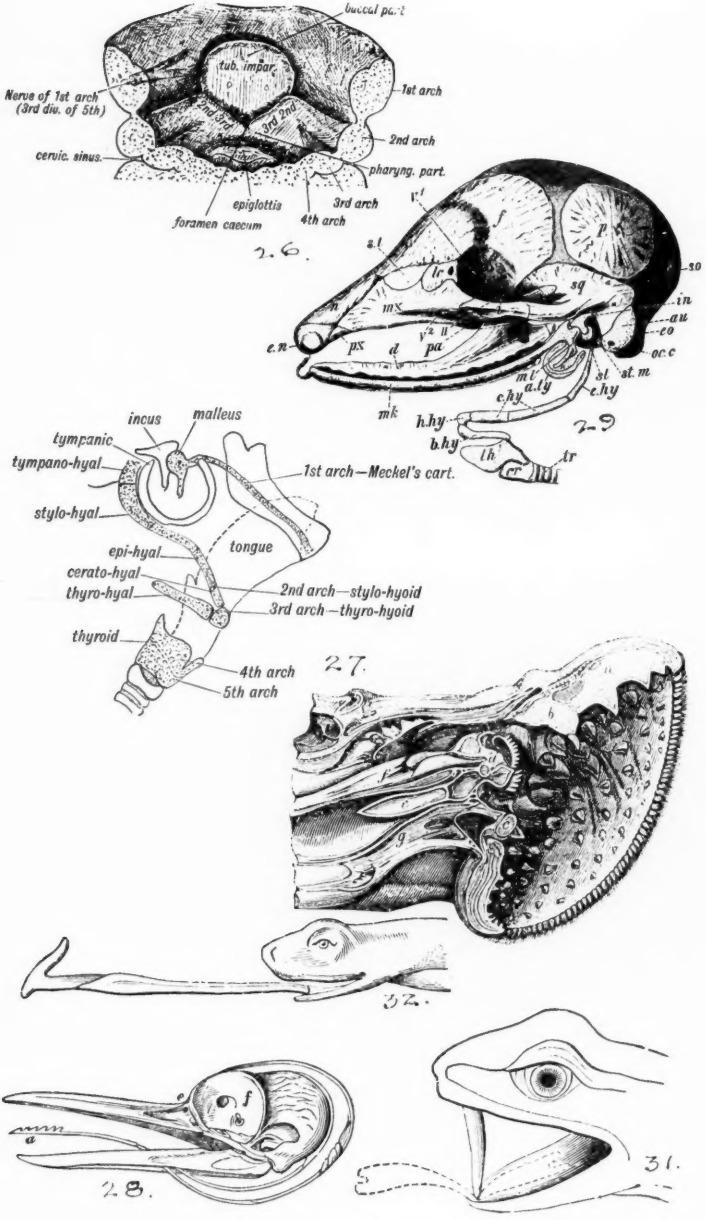
In the humming bird (Fig. 16B) the horny sheath of the glossohyal is divided at its extremity into a pencil of fine hairs. In the Toucan's tongue (Fig. 17) the sheath gives off bristle-like processes from the lateral margins. It becomes an instrument for testing the softness and ripeness of fruit.

In the woodpeckers the apex of the horny sheath (Fig. 28) gives off at the sides, short pointed processes directed backward converting it into a barbed instrument for holding fast the insects, which its sharp point has transfixed after the strong beak has dislodged them from their hiding places.

In the vultures the sides of the tongue can be approximated to form a canal. In the raven it is bifid at the apex. The tongue of the kingfisher (Fig. 16C) is remarkably short, while that of the snipe (Fig. 16A) is remarkable for its length and slenderness.

Parrots have the largest tongue, due to fat, vessels and glands rather than any special muscular development. It serves to keep the nut or kernel steady while being comminuted by the mandible and seems to exercise some gustatory function, since indigestible parts are rejected.

The tongue of birds is controlled by the usual hyodeian muscles, plus four other pairs, two of which are of considerable importance. The cerato-hyoideus passes from the thyrohyal to the urohyal, and is therefore subservient to the lateral movements of the tongue. A


buccal pa.t
tub. impar.
Nerve of 1st arch (3rd div. of 5th)
1st arch
2nd arch
cervic. sinus.
pharyng. part.
epiglottis
3rd arch
foramen caecum
4th arch
26.
so
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h.hy
b.hy
lh
tr
29
incus
malleus
tympanic
tympano-hyal
1st arch—Meckel's cart.
stylo-hyal
epi-hyal
tongue
cerato-hyal
thyro-hyal
2nd arch—stylo-hyoid
3rd arch—thyro-hyoid
thyroid
4th arch
5th arch
27.
32.
28.
31.

singular pair of muscles, the cerato-tracheales (Fig. 18), arise from the trachea, twist four times spirally around the trachea and then pass forward to be inserted into the base of the thyrohyals. This is the principal retractor of the tongue.

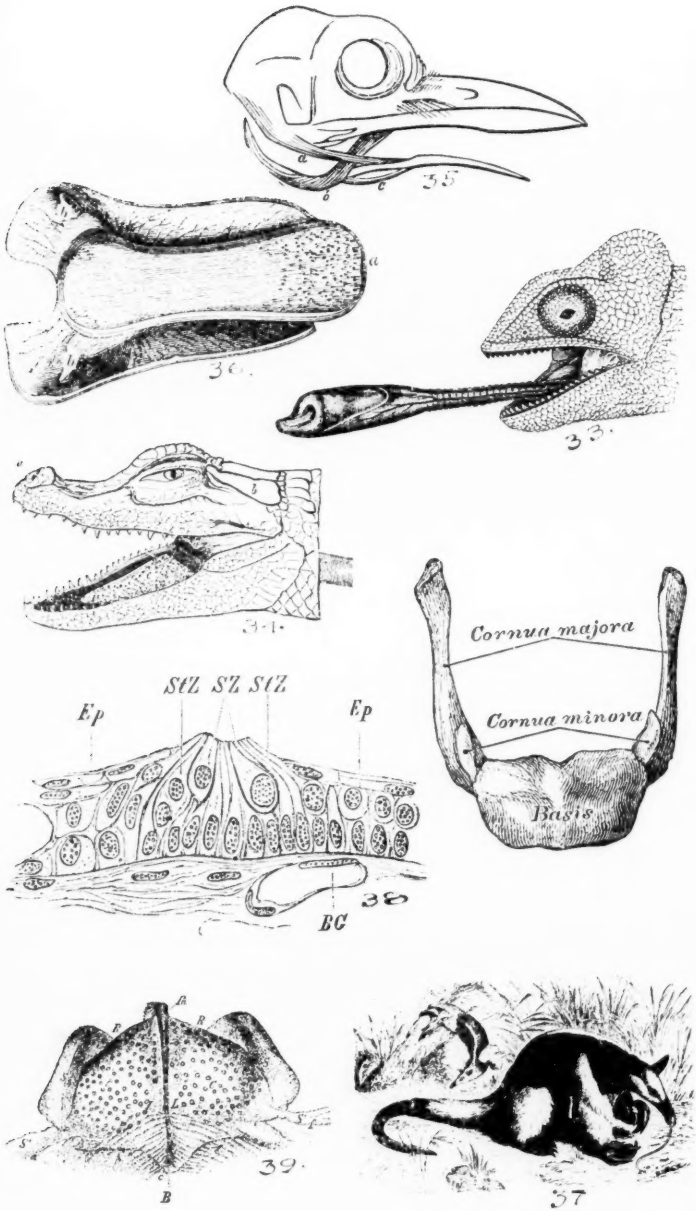
The tongue reaches its most complete morphological and physiological development in mammals and undergoes the most various modifications in form and function. It is, as a rule, flat, band-like and rounded anteriorly and, except in whales, is always very mobile, reaching the extreme in this respect in the anteaters. This mobility is largely due to the intrinsic musculature which is highly developed and may even extend backwards over the sternum. In descending through the mammalian series the mechanical offices of the tongue predominate over the sensitive ones, the form and structure being closely linked with the feeding habits of the particular animal.

In the whale or sea-cow the tongue has little mobility and a small extent of free margin. It is chiefly remarkable for its huge size (Fig. 36), whereas that of the anteater is extremely mobile (Fig. 22) almost like a whip lash, its use being largely to procure its food.

The giraffe (Fig. 19) possesses extraordinary command and power over the movements of its tongue, which is extremely extensible and flexible, and deeply pigmented, probably due to its frequent exposure to a tropical sun. It is able to twine its tongue in a firm but regulated grasp around very small objects. The muscles are the same in kind and number as in other ruminants, the principal difference being in the great extent of the organ anterior to the insertion of the genioglossus, which increases the bulk of the intrinsic as compared to the extrinsic muscles. The intrinsic muscles are less closely interwoven to allow more separated action.

The tongue of the horse (Fig. 20) is relatively long and narrow with a great extent of free tip, with much motor and prehensile power. The root and body of the tongue of the ox (Fig. 21) are wider than that of the horse, but the free part is more pointed. The posterior part of the dorsum forms a remarkable eminence, which is sharply defined in front by a transverse depression. In front of this prominence there are large and horny conical papillæ, with sharp points directed backwards, which impart to the tongue its rasp-like roughness.

The tongue of the hog (Fig. 22) is long and narrow and the apex is thin. The edges of the forehalf are fimbriate. Near the base are two large circumvallate papillæ and it has a double frenum. The


35
36.
33.
31.
Cornua majora
Cornua minora
Basis
StZ SZ StZ
Ep
Ep
38
BC
39.
B
37

dog's tongue (Fig. 24) is wide and thin in front and very mobile. It is deep red in color and the dorsum is marked by a deep median groove. In the inferior part of the tip there is a fusiform cord composed of fibrous tissue, muscular tissue and fat, and in a large dog this may be two inches long. It is called the Lyssa or Sublingua and is supposed to correspond to the last vestige of the tongue of lower vertebrates which has been replaced by the more highly developed organ, characteristic of mammals. A remnant of this can commonly be seen in the under surface of the human tongue, especially just after birth. The tongue of the cat (Fig. 7) is chiefly remarkable for the large number of conical papillæ on the fore part of the dorsum. These are sheathed by horny epithelium, forming retroverted spinules, which impart to the tongue its rasp-like character.

The Tongue as an Organ of Taste. The organ of taste is the only one of the special senses which does not become enclosed in a definite mesodermic capsule. The lowest form of a specialized sense organ is the so-called nerve eminence (Fig. 38) located in the integument of lower animals. This consists of a central group of cells, arranged in the form of a round depressed pyramid composed of sensory cells, surrounded by a network of nerve fibres and supporting cells. These organs have definite locations in the head region and along the sides of the body and tail. They are innervated by branches of the facial, glossopharyngeal and vagus nerves.

In lampreys these special organs remain in a primitive stage of development, but in the common bony fishes they become of great importance and are scattered irregularly over the whole body, but are particularly numerous on the fins, lipfolds and mouth. In the lung fishes they are limited to the oral, pharyngeal and nasal cavities and are supplied by the seventh, ninth and tenth nerves. They are shaped like beakers. In amphibians they occur on the papillæ of the oral and pharyngeal mucous membrane, on the margins of the jaws and on the apices of the fungiform papillæ of the tongue. The latter are probably the first to have a true gustatory function. The taste organs in the frog (Fig. 6) consist of flat disks and occur in hundreds on the tongue and soft palate. The taste cells terminate in a fork with two prongs, or occasionally with three prongs.

In reptiles the distribution of these organs is somewhat more limited and in birds true taste-buds, though present in the mouth and pharynx, are wanting on the horny tongue. The lingual branch of the glossopharyngeal is strongly developed in many groups and functionally replaces the sensory branch of the trigeminal which is wanting in birds.

In all the orders of insects there are found in the tongue, the maxillæ and the mouth, certain minute pits, in each of which is a minute hair or rod which serves as the organ of taste (Fig. 39). These vary greatly in number in different insects. The wasp has 80, a drone 50, the queen bee about 100 and the honey bee about 110.

In mammals organs of taste are still found on the soft palate, on the walls of the pharnyx and even in the larynx, but it is in mammals that the tongue attains its full development as an organ of taste and, as regards the extent and organization of the gustative surface, it is highest in man, due to the enormous number of sensitive papillæ rising above the epithelial level (Fig. 25). These papillæ are either filiform, fungiform or circumvallate, the latter being eight to fifteen in number, forming a V on the dorsum of the tongue, with the apex just in front of the foramen caecum. These are the most important seats of the taste buds (Fig. 5), which lie imbedded in the epithelium lining the groove encircling the central elevation of each papilla. The taste buds are said to number 1,500 in the rabbit, 9,500 in the pig, 9,600 in the sheep, 35,000 in the cow and a much greater number in man, where they almost touch each other in some areas. These papillæ vary tremendously in size, shape, length, texture and number, so that they impart, to the tongue of an animal, characteristics which may serve to differentiate it from the tongue of any other animal.

The human tongue (Fig. 25) mainly differs from that of other mammals in being less massive, less thick relatively to its length and breadth, with a greater proportion of its margin free. It is the most perfect of all tongues in its gustative and other sensibilities and especially in the rapidity, freedom and variety of its movements, whence its applicability to the numerous exigencies of articulate speech as well as to prehension, mastication and deglutition of alimentary substances.

SUMMARY

1. The tongue is the only organ in the body with a double origin, hence its double nerve supply.

2. The ear, and the tongue in part, have a common origin and are linked together by the stylohoid, so have a close association both in birth and in practical everyday use.

3. It starts out in the lower forms as a more or less useless organ, later becoming the chief means of procuring food and ends in man as an organ of taste.

4. The most curious tongues are those of the frog where it flops in and out of the mouth like a door on a hinge, and that of a woodpecker, where the hyoid winds around the head so this bird has a windup like a baseball pitcher.

5. Man is the only animal with a foramen caecum and a chin.

MEDICAL ARTS BLDG.

XVII

IS THE HEARING AID THE PROBLEM OF THE OTOLOGIST OR THE LAYMAN?*

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That the discussion of the deaf individual who is destined to use a hearing device is a timely one becomes most evident when one considers that in the United States today fifteen million people suffer from impaired hearing, about five million of whom are considered deafened enough to require hearing aids. This figure constitutes 4% of the population as actual potential consumers of hearing aids creating a potential market, exclusive of repairs and replacements of over three billion dollars. It is small wonder then that there has been an influx of business men who have sown this fertile field with the seeds of publicity promoting the sale of hearing aids and who have reaped for themselves fat commercial profits.

We, as otologists, have been particularly lax in not heeding the unfortunate mistake of the ophthalmologist in allowing the optometrist to invade his profession, by ourselves permitting a quasi-medico, styling himself as an "audiometrist" or "specialist in hearing aids," to invade our own specialty. I do not, however, feel that the situation is by any means hopeless and will try to analyze it, set forth the necessity for reform, and so far as possible, the means of its accomplishment.

First, the distinctions between the otologist and the "hearing aid specialist" fall into two categories:

1. Differences in the manner of approach to the patient's problem and in the end actually sought to be accomplished;

2. Difference in their background, training and abilities which are reflected in the character of the service which is rendered to the patient both from a mechanical and psychological point of view.

The end which the "hearing aid specialist" seeks to accomplish is to consummate a sale, all other considerations are so far subordi-

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nated as to be practically non-existent. These men are salesmen; skillful, high-powered salesmen. They realize that all deafened individuals, living as they do in a silent world and consequently suffering economically, socially and personally, will make every effort to re-enter the world of sound. This is as true of the intelligent as of the illiterate. The salesman, therefore, plays on the ever tuned strings of human hope by inserting, often without regard for the truth, glowing and promising advertisements in newspapers and periodicals.

It is human to serve one's own interests. Little reflection is necessary to conclude that the "hearing aid specialist's" interests are best served by the sale of a hearing device, if possible one that will actually prove an aid, but in any event a sale must be made.

On the other hand, we otologists have nothing to sell. We are interested in rendering a service rather than in marketing a mechanical device. That service consists in helping the hard of hearing individual by guiding him into those channels, whatever they may be, that will best correct and relieve his failing.

We are interested in his using a hearing aid only when one is indicated and then only from a scientific and not from a self-serving finanical point of view. Considering such duty to our patients and to the public generally, it is lamentable that we have inadvertently overlooked the practical application of the "hearing crutch" or hearing aid. We have kept abreast with the literature, the scientific research, the anatomy, pathology, physiology and etiology concerning impaired hearing and we have instituted and improved the therapeusis indicated, yet, we have overlooked the practical and by no means unimportant problem of hearing aids. We have thereby exposed our patient to treatment by salesmen whose medical scientific background is practically negligible.

We cannot deny that there are many skilled and conscientious hearing aid technicians, some of whom know when to stop. But their number is few, while that of the commercial salesmen is many.

It should not be forgotten that the determination of the device properly applicable to a given case is in itself a scientific problem. How can its solution be properly entrusted to an untrained individual who has memorized a stereotyped "song and dance" which he indiscriminately applies to every individual whether the type of deafness be air, bone, toxic or otherwise.

While it is true that many individuals are helped physically when they purchase a hearing aid from one of the supply houses, such

occurrences are in spite of, rather than because of existing conditions. Then too the psychological approach and the encouragement afforded by the physician, elements of great importance in themselves, are lost entirely.

It seems to me that the facts irrefutably indicate that the proper person to determine whether or not a hearing device is necessary and if so, what its nature should be, is the otologist and not some person who in addition to lacking the necessary training is untrammeled by ethical responsibilites. To this end the profession as a whole should influence legislative action which would make it unlawful for any layman to sell any hearing aid, of any type, except upon a prescription and specification provided by a physician.

To facilitate such prescribing, a standardization of all hearing aids will be necessary. That such standardization is feasible, I will demonstrate later. We as otologists should exert the pressure of our position and demand that in the future all hearing aids on the market be standardized.

If they are, who will be in the better position to prescribe exactly the correct device required for the hearing deficiency in any particular case? Obviously, the otologist with an audiogram could prescribe precisely the hearing deficiency to be corrected and could therefore eliminate the empirical juggling of the apparatus by the salesman.

Upon casual examination the hearing aids appear rather complicated. Upon investigation, however, it is found that the mechanics can be learned in short order. Frankly speaking, if the salesman can handle the hearing aid, why cannot we as otologists do so?

The prerequisites for a good modern hearing device are the following: (1) a microphone; (2) a battery; (3) an amplifier; (4) a receiver.

The receiver may be an air or a bone conductor, or a combination of both. The microphone picks up the sound wave and converts the motivating power of the battery into electrical pulsations which are carried to and through the receiver. The amplifier intensifies the electrical pulsations and ultimately the sound output. The rheostat controls the output volume. Our object therefore is to assemble these different parts mentioned and if the combination is thoroughly coördinated mechanically and electrically with proper adjustment to the patient—that patient will have a hearing aid suitable to his or her type of deafness.

There are certain physical laws of sound and their relationship to the human ear with which we must reckon.

Hearing is a normal physiologic phenomenon; it is the perception of vibrations from an acoustic medium. This medium may be the human voice, the telephone, a musical instrument, noise, tuning fork, watch, audiometer, and the like.

While the human ear is best adapted to the human voice, modern civilization with its complexity of noises and sounds has found that it has become the receptor of many sounds wholly different in character from that of the voice. Hearing is a subjective phenomenon. Many elements come into play, such as physiologic variations, anatomic anomalies, age, intelligence, volume, tactility of sound and special ear training.

The human ear can readily distinguish different frequencies. The scale of tones appreciated by the human ear is divided into octaves for testing purposes. Loudness or intensified sound is difficult for the human ear to differentiate, as intensified sound does not register in pitch variations. The human ear is so constructed that it is able to recognize high, medium and low tones. The hearing range is approximately between 30 and 12,000 cycles or double vibrations per second. The hearing fields must be investigated from the minimal possible perception of sound to the maximum. Human conversation has its optimum acuity between 300 and 2500 cycles per second.

Sound is transmitted to the ear by air or bone or a combination of both. Hearing aids must therefore supply sounds for either air or bone conduction, or both as required by the patient.

As previously stated the human ear is best adapted to the human voice. Early hearing tests were purely subjective and consisted of the whisper, spoken and high pitched voice tests. These tests were of value only to the otologist as a personal record. Therefore otologists sought some standardized mechanical means, such as the tuning forks, monochord and audiometers, to chart the acoustic fields. Audiometer and tuning fork tests attempt to measure tone perception quantitatively as well as qualitatively. In order to calibrate the physical insufficiencies of the acoustic range we therefore use the standardized means for testing purposes.

Since hearing is perceived by air and bone conduction or a combination of both we must not lose sight of the fact that disease may affect both air and bone conduction or destroy partially or completely either air or bone conduction. Various diseases affecting the ear may

partially or completely destroy perception for any one of the sound levels. Therefore, by searching the human hearing range, one may find isolated islands of sound which can be utilized for hearing aids.

It is of paramount importance that the hearing aid be adapted to the type of deafness present. For example, in mild cases, an individual may hear the voice and sound in various pitches, but in conversation particularly, the sounds may be so muffled that they do not register intelligibly. This individual would not need to be supplied with a particular pitch, but would require amplification. In other words, the amount of amplification would have to vary with the loss in the different frequencies and be so fixed as to raise the individual's threshold of hearing.

If one has a great hearing loss of any one of the tone ranges, the hearing aid should emphasize sounds of that particular range which is missing or deficient.

Normally we hear best by air conduction. Therefore, when we have a loss of air conduction and the residual air conduction is still better than bone conduction our prothesis should be adapted and concentrated upon air conduction. On the other hand if bone conduction is the predominant element then we must take advantage of this medium and the individual should be supplied with a bone conductor.

For those who are bilaterally deaf, and have made up their minds to wear hearing aids, separate instruments for each ear can be worn and the hearing deficiency may be corrected according to the type of hearing loss.

In making a tour of the various hearing aid shops, including the department stores, optical places, et cetera, I learned that most manufacturers make microphones which are adjusted to the three intensity levels: low, medium and high. They also supply several types of air and bone conducting units. These units are assembled and tried with the various microphones and amplifiers empirically and a selection is made by the deaf individual after tests and various combinations of these units have been tried.

Other manufacturers make an entirely different type of hearing aid. They adjust various carbon plates in the amplifying unit. These carbon plates, in addition to the air and bone conductor, are juggled around in the amplifying units, with the patient as the subject, until the maximum hearing is obtained.

The selective type of hearing aid shows an improvement upon the old type of instrument.

Newhart and Hartig[2] have found that all hearing aids are not accurately calibrated and that there is a great deal of variation qualitatively as well as quantitatively.

I might mention that individuals wearing hearing aids should be brought back at various intervals for minor corrections and adjustments; new equipment or replacements may be necessary from time to time. It is also essential to study the reaction of the individual and determine whether he has conquered the instrument or has been conquered by it.

The deaf individual still regards the wearing of a hearing aid as a stigma. Therefore, the deafened as well as the hearing public should be educated to its necessity, just as years ago, the public was educated to the wearing of spectacles.

COMMENT

1. A line beween the deaf and the hard of hearing should be drawn.

2. It is the duty of the otologist to seek the cause of the deafness and alleviate any direct or remote causes.

3. All possible foci of infection should be removed and all known therapeusis should be instituted.

4. The national oto-rhino-laryngological societies should at their sectional meetings inform their memberships of the simplicity of the hearing aids and the manner in which they are handled.

5. Special instructional courses should be given at the sectional meetings of the national organizations.

6. In order to combat the quasi-medico, every otologist should acquaint himself with every possible type of hearing aid. He should collect the entire paraphernalia, including the various microphones, the rheostats, batteries, amplifiers, receivers and selectors, if necessary.

7. Just as it is important for hearing tests to be standardized, so it is for hearing aids to be standardized.

8. The otologist should by all means do the testing and he should determine what type of artificial contrivance is best suited for the individual.

9. The lay hearing-aid technician may understand the physics of sound but he is entirely deficient in the knowledge of the anatomy, physiology, pathology, etiology, treatment and especially the psychology of the deaf.

10. The object of the hearing aid is to secure better hearing for the individual, prevent exhaustion of the cochlear nerve, prevent mental strain, improve the general well being of the patient, help him mix with normal people and last but not least, help improve his social and economic status.

11. The profession as a whole should influence legislative action and make it compulsory that no lay person be permitted to sell a hearing aid without the physician's prescription and specification.

1915 SPRUCE STREET.

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XVIII

BLOOD CELL RESPONSE TO SULFANILAMIDE THERAPY*

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CHICAGO

Although sulfanilamide is being very widely used, very little is known of its mode of action. Symptoms of toxicity such as skin rashes, nausea and vomiting, dizziness and hyperpyrexia have been reported during administration of the drug. Cyanosis during therapy has also been reported. So far, only relatively few cases of blood cell depression have been reported due to its use, although there is an undercurrent of feeling that there may occur the same marked depression of the blood elements as is being reported with such drugs as amidopyrine.

Mossell[1] reports the case of a child with rheumatic fever treated with sulfanilamide. During therapy the white blood cells reached a low level of 3,700, but upon stopping the drug they increased to 8,000 to 10,000. The case of a woman, aged 54 years, with a streptococcus septicemia is reported by Plumer.[2] Before sulfanilamide therapy, there was a leucocytosis of 26,000. The drug was given for five weeks, after which it was stopped because of nausea and vomiting. After the fourth week of therapy the leucocyte count was 21,000. Twelve days later, which was three days after the discontinuance of medication, the white cells had decreased to 400 with no polymorphonuclear neutrophils. By the following day the leucocytes had increased to 16,000.

The following two cases of leucopenia are reported by Trumper:[3] An adult with a septic mastoiditis had a leucocytosis of 18,000 with 85 per cent polymorphonuclear neutrophils before sulfanilamide therapy. During therapy the leucocytes decreased to 4,000 with 35 per cent polymorphonuclear neutrophils. After the drug was discontinued the white cells and the polymorphonuclear neutrophils increased to their former high levels. The second case was a primipara with a post-partum fever. The leucocyte count was 20,000 with over 80

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From the Otho S. A. Sprague Mem. Institute and The Children's Memorial Hospital.

per cent polymorphonuclear neutrophils before the drug was given. During therapy the leucocytes decreased to 6,000 with 50 to 60 per cent neutrophils. After therapy the white cells increased to 13,000 with 80 per cent neutrophils.

Borst[4] reports the case of a woman, age 61 years, who was being treated for a pyelocystitis. On admission the leucocyte count was 8,000 with 64 per cent neutrophils. Sulfanilamide was given for slightly longer than four weeks. Early during the administration of the drug the leucocyte count was 14,000 with 90 per cent neutrophils. On the day the drug was stopped, which was two days before death, the leucocyte count was 1,125 with 85 per cent lymphocytes, and on the following day the count had decreased to 960 with 87 per cent lymphocytes. In this patient there was a history of vaginal bleeding, bleeding from the gums and petechiæ of the skin ten years previously, and six years previously petechiæ of the skin. In view of these facts one might question whether the agranulocytosis was not due to a blood dyscrasia or reaction to the infection rather than to the drug. A case of fatal agranulocytosis is reported by Young.[5] This occurred in a man being treated for an acute rheumatic infection. Sulfanilamide was administered for eighteen days. At the beginning of therapy the leucocyte count was 12,000, of which 4,344 were lymphocytes. There was a gradual reduction of the leucocytes to 7,800, of which 1,755 were lymphocytes, on the day before the drug was stopped. Five days later, the day before death, there were only 1,800 leucocytes, all of which were lymphocytes. Necropsy: Death due to agranulocytic angina with hemolytic staphylococcus aureus and streptococcus viridins septicemia.

Harvey and Janeway[6] report three cases of a hemolytic type of anemia and mention three other similar cases which occurred during sulfanilamide therapy.

As yet the literature contains no reports of daily blood counts during sulfanilamide therapy. With the idea of determining the leucocyte response in patients receiving sulfanilamide, we made daily blood counts and Schilling differential counts before, during and after such therapy on a group of 33 patients. The leucocyte counts were made at approximately the same hour of the day. A dosage of 15 grains of the drug per 20 pounds of body weight was very closely adhered to. Patients with various types of infections as well as those with no apparent infection were included in the study.

Of the 33 patients, 27 had a leucocytosis and six had a leucocyte count within normal limits when sulfanilamide therapy was started.

In both groups there were patients with infection and fever, infection without fever (usually chronic infection) and with no evident infection.

RESPONSE WHEN LEUCOCYTOSIS WAS PRESENT

A. Infection with fever was present in 17 of the patients with a leucocytosis at the time therapy was started.

In seven of the patients the leucocytosis decreased to normal in a crisis-like manner within 24 hours after the fever had subsided. This occurred after from one to four days of medication. In most instances, after sulfanilamide was discontinued, there would occur a moderate increase in the leucocytes for a few days, after which they would decrease to a normal level again. If there was a recurrence of the infection a leucocytosis again would develop. Upon readministration of the drug the same crisis-like drop in white cells occurred upon subsidence of the fever. In this group there were four cases of otitis media and mastoiditis, one otitis media, one otitis media with erysipelas, and one erysipelas, all with positive hemolytic streptococci cultures (see Charts 1, 2, 3).

The leucocytes decreased in a lysis-like manner in from three to five days after subsidence of the fever in four patients. There was again a moderate increase in the leucocytes for a few days after medication was discontinued. The gradual decrease in leucocytes to normal was similar to the leucocyte response in infections when sulfanilamide is not used. In this group were two cases of otitis media and mastoiditis, one with erysipelas and one with a septic temperature following a pneumonia. In only the last case was a positive hemolytic streptococcus culture not obtained (see Charts 4 and 5).

In six critically ill children who received sulfanilamide for only a short period, there was no change in the leucocytosis during therapy. In this group the drug was discontinued before recovery from the infection because of the occurrence of reactions. The total leucocytes neither increased nor decreased other than by minor fluctuations which could be accounted for by the course of the infection. There is no question but that the sulfanilamide played an important part in the recovery of these patients, but there was no evidence that the drug brought about the aid through the white blood cells. In this group of six cases one was a hemolytic streptococcus peritonitis, one a hemolytic streptococcus septicemia and hip abscess, one a meningococcus meningitis, one a hemolytic streptococcus infection of the mastoid with erysipelas, one an erysipelas and abscess of the leg (hemolytic streptococcus), and one a hemolytic streptococcus infection of the hip. All of the patients recovered, and in the two cases

with erysipelas there was a very prompt recovery of that part of the infection (see Chart 6).

B. There were three patients with a leucocytosis in whom an infection without fever was present.

The leucocytes decreased to normal in a crisis-like manner in two patients. Both of these patients had a subacute suppurative otitis media due to a hemolytic streptococcus infection. In one the ear stopped discharging within 24 hours after sulfanilamide was started, and in the other after three days' therapy with the drug. In both the leucocyte count reached normal on the days the ears stopped draining. In both children a few days after therapy was discontinued there was a recurrence of the discharge from the ears and a moderate increase in the leucocytes, but when sulfanilamide was again given the ear discharge immediately ceased and the leucocytes promptly returned to a normal level within 24 hours. The leucocyte count decreased to normal over a four-day period in one case. This was in a patient with an otitis media and a subperiosteal abscess which ruptured spontaneously. It was a hemolytic streptococcus infection which healed by the fourth day of medication.

C. A leucocytosis was present in seven patients in whom there was no evident infection, but four of them had recently recovered from a throat infection.

A crisis-like drop of the leucocytes to normal occurred within 24 to 36 hours after medication was started in four patients. All of them had a persistently positive throat culture for hemolytic streptococci. There was a leucocytosis of from 12,000 to 14,000 before sulfanilamide was given. In all of the cases there was again a moderate increase in the leucocytes for a few days after medication was discontinued. The positive throat cultures became negative and remained so for several days, after which they all became positive again (see Chart 7).

A lysis-like decrease in the leucocytes from 14,000 to 16,000 to a normal level occurred in two patients. This decrease took place over a period of three days during sulfanilamide medication. Why the leucocytosis was originally present, we do not know, but white blood cell counts were made for four consecutive days before medication was started and they continued elevated during this period, only decreasing to a normal level with administration of the drug.

In one patient with a leucocytosis of between 8,000 and 10,000, without any evident infection, there was no change in the white cells after five days of medication.

RESPONSE WHEN A NORMAL LEUCOCYTE COUNT WAS PRESENT

There were six children in this group that received sulfanilamide.

A. Infection with fever. The one patient in this group had a severe tonsillitis and ulcerative stomatitis. Cultures and smears showed fusiform bacilli and spirillæ. The leucocyte count was 7,000 before sulfanilamide was given. The drug was given for two days and then had to be discontinued because of vomiting and a severe acidosis. The leucocyte count remained between 7,000 and 9,000 during therapy and until the infection cleared up. Sulfanilamide had no therapeutic effect in this infection.

B. Infection without fever was present in three children. In two of these patients with a white cell count of from 7,000 to 8,000 before therapy, a mild leucopenia without neutropenia developed after from three to four days of sulfanilamide therapy. The leucocyte count decreased to 4,800 in both patients and in one of the patients this occurred on two occasions when the drug was given. Within a few days after therapy the white cells again increased to a normal level. Both of these children had a chronic otitis media from which the proteus bacillus was cultured. There was no effect on the discharge or on the cultures. These were the only two patients in which a leucopenia developed with sulfanilamide medication (see Chart 8).

The leucocyte count showed no change from the normal in one patient with a chronic otitis media from which the proteus bacillus was cultured. Before, during and after sulfanilamide therapy, the white cell count was 8,000. The ear discharge and infecting organism were not affected by therapy.

C. No infection was present in two patients with normal white cell counts that received sulfanilamide. In both of these cases there was no change in the leucocyte count during or after therapy. The white cells remained between 6,500 and 8,000, the same level as had been present before therapy (see Chart 9).

COMMENT

White Cells. Not including the six patients to whom the drug was not administered until the infection subsided, there were 21 cases with a leucocytosis. In 13 of these 21 cases the leucocytes decreased to normal in a crisis-like manner within 24 to 36 hours after the subsidence of the fever. In seven cases the white cells returned to a normal level in a lysis-like manner only after 48 hours or more, while in only one case the leucocytosis did not change.

Of six cases with a normal white cell count to whom the drug was administered, a moderate leucopenia without neutropenia developed in two. In the other four the leucocyte count did not change.

The alteration in the leucocyte response was in the nature of a marked absolute reduction of all the cell elements without any characteristic relative change in the proportion of the different white blood cells as studied by the Schilling count. The granulocytes were not reduced out of proportion to the other cells and at no time during the administration of sulfanilamide could toxic changes be found that had not previously been present as a result of the infection alone. In the majority of cases the Schilling differential count reflected only what one could consider a healing infection.

An increase in the leucocyte count which could be attributed to the sulfanilamide did not occur in a single case. On the other hand, it did seem as if the drug did cause a depression of the white blood cells. This is borne out not only by the two cases of leucopenia that developed, but by the spectacularly rapid decrease in the leucocytes as the infection subsided, which was often then followed by a moderate leucocytosis after the drug was discontinued. Too, in six out of seven patients without an infection but with a leucocytosis, there was a rapid decrease of the white cells to a normal level when the drug was administered. From this it would seem that the beneficial action of sulfanilamide is due to its own action in the blood stream rather than to its enhancing the power of the leucocytes or increasing their number to control the infection.

Red Cells. No change in the number of red blood cells occurred other than could be expected during the course of the infection in this series of cases.

Blood platelets. We observed no changes in the number of platelets during or after therapy.

Hemoglobin. Hemoglobin determinations were made by the Sahli method. During therapy there was little decrease in the hemoglobin by this method of determination; in fact, not as much as one would expect during the course of infection, and it was not unusual to find the hemoglobin increased. Cyanosis developed in seven of the patients (two of them in normal children being given the drug) but it never lasted for more than three to four days. This same high frequency of cyanosis occurred in the other patients treated at the hospital but not included in the above study. When cyanosis was present, or even before it occurred, a brownish color of the blood was easily detectable. The hemoglobin (Sahli) would usually be 90 to

100 per cent before the cyanosis developed, and during the period of cyanosis levels as high as 138 per cent were reached with a gradual decrease as the cyanosis disappeared. In none of the above cases of cyanosis was a methemoglobin or a sulfhemoglobin band demonstrable on blood specimens with the hand spectroscope. Several patients were again given the drug after the cyanosis had disappeared and in only a few of them did it recur. It seems from the above that sulfanilamide produces some changes in the already existing blood pigments or possibly by chemical action a new pigment relating to sulphur is formed which produces changes in the color of the blood so that colorometric determinations are not accurate for hemoglobin determinations in these patients.

RESPONSE OF INFECTIONS

Careful observations were made on the 93 patients treated with sulfanilamide included in Table I. The cases are listed according to the type of infection and the infecting organism when known. In considering whether the response to sulfanilamide therapy was favorable, questionable, or whether the infection ran its normal course, there is, of course, the personal equation of the interpreter to consider. Insofar as possible a conservative attitude was taken. Consideration was not only given to the individual case being treated but also to similar cases being observed at the same time but not receiving sulfanilamide therapy. For instance, if an otitis media with profuse suppuration of one or two days' duration stopped discharging after 24 to 48 hours of therapy and the patient remained well, then that case was listed as a favorable response. If the acute symptoms of pain and fever disappeared immediately with therapy and the discharge gradually stopped over a period of a week or ten days, then the response was considered questionable. If there was no abatement of symptoms or discharge then it was considered that therapy was of no benefit. When the results of the cases are listed on the same line with the diagnosis the infecting organism was not known.

Undoubtedly all the cases included in the mastoiditis group did not have a true mastoiditis, although severe mastoid tenderness and pain were present with edema in some instances. In one of this group with a favorable result, a subperiosteal abscess over the mastoid ruptured spontaneously with complete healing and cessation of the discharge from the ear in four days. Four of the eight cases of hemolytic streptococcus mastoiditis listed as questionable responses were operated upon and one was not. The wounds healed and the ears became dry in what was considered somewhat better than average

time. In the other three cases of this group mastoid tenderness disappeared and the ear discharge stopped with therapy but recurred again within a short period after therapy was stopped. Both of these cases, it was felt, had to be operated with the recurrence of symptoms and the course of recovery was as short, if not shorter, than one would normally expect. Three cases of mastoiditis and otitis media with a positive staphylococcus aureus culture quieted down after sulfanilamide therapy and one case had to be operated.

Of the infections included in the miscellaneous group, two were cases of cervical adenitis and throat infections in which favorable responses were obtained, while two rheumatic infections, an infected burn, a retrotonsillar abscess, a stomatitis, a pneumococcus empyema and a staphylococcus aureus abscess showed no response to therapy. In one case of pneumonia the response to therapy was questionable.

It can be readily seen that the streptococcus infections show by far the best response to sulfanilamide therapy. When an abscess has formed or when bone destruction is present even though the infecting organism is the hemolytic streptococcus, the results of therapy are less spectacular. Usually, then, surgery must be resorted to. Six of the seven cases that had persistently positive hemolytic streptococcus throat cultures cleared up for several days when sulfanilamide was administered, but after discontinuance of the drug they all became positive again.

CONCLUSIONS

1. Sulfanilamide is an effective therapeutic agent in most hemolytic streptococcus infections.

2. Cyanosis is of frequent occurrence during administration of the drug.

3. There is evidence that sulfanilamide has a tendency to cause a depression of the leucocytes.

4. The action of sulfanilamide seems to be independent of the leucocytes in that it does not produce an increase in the total leucocytes or in the proportion of the polymorphonuclear cells.

5. Frequent blood cell determinations should accompany sulfanilamide therapy.

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Since this study was completed, we have had two more cases of leucopenia develop during sulfanilamide therapy. In both cases infection, fever and a leucocytosis had been present. As the infection subsided the leucocyte count decreased to 5,000 in one and to 5,700 in the other without neutropenia in either case.

CHART I

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
5/3	15 gr.	25,800	....	....	....	....	33	54	87	13	....	13	..	Temp. 105°
5/4	15 gr.	32,450	....	....	....	....	16	58	73	23	....	23	4	Temp. 100°
5/5	15 gr.	6,750	....	1	....	....	11	50	61	20	1	21	17	Temp. Normal
5/6		7,525	....	....	....	....	19	29	48	47	....	47	5	
5/7		11,200	1	2	....	....	17	33	50	32	3	34	12	
5/8		8,900	....	1	....	....	9	36	45	47	....	47	7	
5/9		13,000	....	1	....	....	18	27	45	43	2	45	9	
5/10		8,075	....	....	....	....	20	28	48	45	....	45	7	
5/17		9,600	....	....	....	....	12	24	36	52	....	52	12	

Age 1 year. Otitis media and erysipelas of scalp. High fever for 4 days. Throat positive for hemolytic streptococcus.

CHART II

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
5/6			....	....	....	....	....	....	90	6	....	6	4	Temp. 102.4°
5/7	25 gr.	40,100	....	....	....	5	30	52	87	8	....	8	5	Temp. 104.4°
5/8	25 gr.	15,200	....	....	....	....	32	60	92	6	....	6	2	Temp. 106.6°
5/9	25 gr.	10,200	....	....	....	2	20	35	57	28	....	28	15	Temp. Normal
5/10	25 gr.	6,075	....	1	....	2	18	35	35	30	2	32	14	Rt. Ear Normal
5/11	25 gr.	7,800	....	1	....	....	29	33	62	26	2	28	10	Mastoid Normal
5/12	25 gr.	7,400	....	1	....	....	19	39	58	22	....	22	19	
5/13		8,450	1	1	....	....	25	39	64	24	....	24	10	Slight Discharge
5/14		7,150	....	2	....	....	14	42	56	31	2	33	11	From Left Ear 5/22

Age 6 years. Bilat. otitis media, left mastoid tenderness and swelling. Fever for 5 days. Throat positive for hemolytic streptococcus.

CHART III

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/24		21,650	....	2	....	....	23	47	70	23	....	23	5	Temp. 101.4°
4/25		20,700	....	2	....	....	24	56	80	12	....	12	5	Temp. 100.6°
4/26	30 gr.	20,000	....	2	....	1	26	50	77	16	....	16	5	Temp. 101°
4/27	30 gr.	13,325	....	....	1	....	5	49	55	40	....	40	5	Normal
4/28	30 gr.	8,850	....	....	....	....	5	55	60	30	....	30	10	Mastoid O. K.
4/29	30 gr.	10,950	....	....	....	....	8	57	65	27	....	27	8	
4/30		10,850	....	....	....	....	12	56	68	30	....	30	2	
5/1		9,425	....	....	....	....	10	49	59	32	....	32	6	Home. Ear Dry
5/4	40 gr.	20,400	....	....	....	....	17	63	80	16	....	16	4	102° Mastoiditis
5/5	40 gr.	10,550	....	....	....	....	8	63	71	18	....	18	10	99.4°
5/6	40 gr.	6,800	....	....	....	....	9	60	69	18	....	18	13	Normal
5/8		7,850	....	....	....	1	14	52	67	30	....	30	3	
5/9		15,200	....	....	....	....	10	58	68	29	....	29	3	6/10 Ear and
5/10		15,300	....	....	2	....	7	53	60	28	....	28	10	Mastoid Dry

Age 7 years. Left sup. otitis media with mastoid tenderness. Ear opened 4/20. Fever for nine days. Readmitted 5/4 with fever, left sup. otitis media and mastoid tenderness. Mastoidectomy 5/7. Ears positive for hemolytic streptococcus.

CHART IV

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
6/2		15,300	—	1	—	—			68	28	—	28	2	Temp. 104.2°
6/4		21,600	—	—	—	—			83	16	—	16	1	Temp. 106°
6/6	Prontosil 20 cc.													Temp. 106°
6/7	Prontosil 15 cc.	16,700	1	1	—	—			76	16	—	16	6	Temp. 105.4°
6/8	45 gr.	12,500	—	—	—	—			71	28	—	28	1	Temp. 105.4°
6/9	45 gr.	10,200	2	2	—	—			67	25	—	25	4	Temp. 105° and Chill
6/10	45 gr.	19,600	—	1	—	—			62	32	—	32	5	Normal Hereafter
6/11	45 gr.	15,700	—	1	—	—			64	31	—	31	4	
6/12	45 gr.	12,700	—	1	—	—			57	36	—	36	6	
6/13	45 gr.	9,100	—	—	—	—			59	37	—	37	3	
6/14	30 gr.	7,600	—	1	—	—			58	38	—	38	3	
6/15		9,700	—	—	—	—			48	52	—	52	—	

Age 9 years. Pneumonia 5 weeks ago. Daily chill and high fever for five days.

CHART V

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/17		19,200	....	....	....	....			44	....	....	44	12	Temp. 101°-10° to 4/2
4/23	20 gr.	25,600	....	....	....	....	16	67	83	14	....	14	3	Temp. 104°
4/24	20 gr.	16,700	....	....	....	1	21	45	67	33	....	33	....	Normal
4/25	20 gr.	14,250	....	3	....	....	18	35	56	40	....	40	4	Infection better
4/26	20 gr.	12,600	....	....	....	1	20	28	49	41	3	44	7	
4/27	20 gr.	8,700	....	2	....	....	14	28	42	46	....	46	10	
4/28		8,000	....	1	....	....	16	17	33	55	2	57	9	
4/29		11,500	....	....	....	....	13	36	49	46	....	46	5	
4/30		12,900	....	....	....	1	14	35	50	42	....	42	8	
5/1		10,525	....	....	....	....	20	48	68	30	....	30	2	

Age 1 year. Infected laceration of eye lid with erysipelas of face, and fever for four days. Wound and throat positive for hemolytic streptococcus.

CHART VI

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/2		21,300	....	....	....	....	67	15	82	12	....	12	6	Temp. 105° Daily
4/5		18,650	....	....	....	....	67	15	82	12	....	12	6	to 4/15; then
4/6		15,850	....	....	....	2	57	20	79	19	....	19	2	103+° 4/27
4/7		18,500	....	....	....	1	51	28	80	17	....	17	3	
4/8		30,000	....	....	1	2	63	17	83	15	....	15	2	
4/9	45 gr.	24,700	....	....	....	6	45	26	77	14	....	14	9	
4/10	45 gr.	20,700	....	....	....	2	68	16	86	6	....	6	8	
4/11	45 gr. thru	17,100	....	....	....	1	36	32	69	19	....	19	12	
4/13	4/13	12,400	....	1	....	5	35	43	84	10	....	10	6	
4/16			....	....	....	2	35	43	80	18	....	18	2	5/25 Rt. hip
4/23	45 gr. from	32,600	....	....	....	....	11	58	69	19	....	19	12	drain Hemolytic
4/24	4/22 to 5/3	23,300	....	....	....	1	27	43	71	19	....	19	10	strep. culture
4/26		17,250	....	....	....	....	15	51	66	25	....	25	9	positive. Temp.
4/27		12,950	....	1	....	....	13	53	66	23	....	23	7	normal after
5/3		16,025	....	....	....	....	15	60	75	21	....	21	4	6/13

Age 8 years. Hemolytic streptococcus infection right hip with septicemia, pneumonia and jaundice. High fever and occasional chill for two weeks. Blood positive for hemolytic streptococcus.

CHART VII

Date	daily dose Sulfanilamide	per cmm. Leucocytes	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/19		12,500	....	9	....	....	13	52	65	13	....	....	13	Temp. Normal
4/20		11,600	1	3	....	....	8	42	50	36	....	....	10	Throat Clear
4/21	60 gr.	11,550,	....	2	....	....	12	49	61	30	....	....	7	
4/22	60 gr.	8,275	....	4	....	....	8	49	57	22	....	....	17	
4/23	60 gr.	9,125	....	2	....	....	17	56	73	21	....	....	4	
4/24		9,600	....	....	....	....	17	63	80	17	....	....	3	
4/25		9,000	....	....	....	....	12	44	56	32	....	....	12	

Age 22 years. Persistently positive throat culture. Had been on and off duty with positive culture for Hem. strep. since 12/2/36.

CHART VIII

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/12		7,400	1	2	....	1	10	34	45	38	....	38	14	No change in
4/13		7,200	....	....	....	....	6	42	48	36	....	36	16	discharge from
4/14		7,600	....	4	....	....	16	40	56	19	1	20	20	ears throughout
4/16	40 gr. 4/15	7,550	1	2	....	....	18	35	53	17	....	17	27	study
4/17	thru 4/18	5,650	....	1	....	....	16	34	50	31	....	31	18	
4/19		4,850	....	....	....	1	18	39	58	20	....	20	22	
4/21		4,950	....	....	....	....	11	33	45	45	....	45	11	
4/24		6,450	....	....	....	1	19	35	55	33	....	33	12	

Age 10 years. Chronic otitis media. Nose, throat and ears negative for hemolytic streptococcus. Ears: Proteus B.

CHART IX

Date	Sulfanilamide daily dose	Leucocytes per cmm.	Schilling Count in Percent Neutrophils B	E	M	J	Stab.	Seg.	Total	Lymphocytes Mature	Blasts	Total	Monos.	Remarks
4/24		7,800	....	....	....	4	17	64	85	5	....	5	10	Throat still
4/26		8,100	....	....	....	....	20	68	88	4	....	4	8	moderately red
4/27	60 gr.	7,700	....	6	....	....	13	50	63	16	....	16	15	
4/28	60 gr.	7,175	....	2	....	....	10	58	68	23	1	24	6	
4/29	25 gr.	8,225	....	....	1	1	15	62	79	16	2	18	3	
4/30		6,625	....	1	....	....	14	56	70	27	2	27	2	
5/1		6,800	....	1	....	....	7	58	65	31	....	31	3	
5/3		7,175	....	1	....	....	7	65	72	22	2	24	3	
5/4		7,900	....	1	....	....	11	66	77	20	....	20	2	
5/5		7,400	....	....	....	....	5	74	79	16	....	16	5	
5/6		5,500	....	....	....	....	10	53	63	23	....	23	10	
5/11		6,150	....	....	....	1	6	58	65	28	....	28	7	

Age 24 years. Throat infection three days ago. Hemolytic streptococcus carrier.

TABLE I

Responses of Infections	Favorable	Questionable	None
Otitis Media	7	2	3
Strept. Hem.	12	2	—
Strept. Viridans	—	—	1
Proteus B	—	—	6
Otitis and Mastoiditis	—	1	—
Strept. Hem.	8	8	—
Staph. Aureus or Albus	—	2	2
Erysipelas	5	—	—
Strept. Hem. septicemia	2	—	—
Strept. Hem. carriers	—	—	7
Osteomyelitis			
Strept. Hem.	—	—	1
Staph. Aureus	—	—	2
Pyuria	3	—	1
Strept. Hem. peritonitis	—	1	—
Meningitis			
Meningococcus	—	2	—
Pneumococcus type IV	—	—	2
Hemolytic Streptococcus	—	—	1
Hemolytic Influenza	—	1	—
Sepsis	1	—	—
Miscellaneous Infections	2	1	7

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XIX

A NEW OPERATION FOR THE CORRECTION OF EXTERNAL DEVIATIONS OF THE LOWER END OF THE NOSE

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Deformity of the cartilaginous septum is a frequent occurrence following trauma to the nose. The pathological forms which the septum may take are the following:

1) Dislocations of the septum, in which the septum in its entirety, is shifted from its normal mid-line position.

2) Fracture-deviations of the septum, in which the septum is broken.

Fracture dislocations and deviations of the septum are always followed by some change in the relative size of the intranasal air passages. If the deviation is marked there may be respiratory obstruction on one or both sides.

External deviations of the lower half of the nose do not always occur when the septum is fractured and deviated. When they do occur they constitute cosmetic disfigurements.

The satisfactory correction of external deviations of the lower half of the nose is complicated by the fact that the cartilage of the septum, like cartilage elsewhere, does not reunite to itself by a chondroplastic process similar to the osteoplastic process that occurs in the formation of callus after bony fractures. The union that takes place is fibrous in character, a type of healing, which, while sufficient to close defects, is not sturdy enough to support the weight of the nose. For this reason, refracturing of the septum, and splinting in the proper position, a process well known and understood in the treatment of bony malunion, is not feasible. Hence, once the septum has been broken, it no longer can be rendered as useful to support the original nose. The nose must either be changed to conform with the new structure of the septum or the septum must be supported by the surrounding structures much in the fashion that an insecure mast can be guyed by ropes or cables.

Fig. 1. A new operation for correction of external deviation of the nose.

It is on the principle of this guying that the operation to be described has been based.

When an external deviation of the lower half of the septum occurs, there is an associated asymmetry of the triangular cartilages (upper lateral cartilages, see Fig. 2). The triangular cartilage opposite the direction of the deviation will be larger than the other cartilage. This asymmetry is probably due to the fact that at the time of the septal fracture, the triangular cartilage was ruptured either from its attachment to the septal cartilage or from its attachment to the frontal process of the superior maxilla or nasal bone. In either case the defect will be closed by a proliferation of scar tissue that renders this cartilage larger than the opposite one.

Any operation that does not include a readjustment of the triangular cartilages will be generally unsatisfactory. An examination of the literature has failed to reveal mention of this fact, and herein the technique to be described offers a new procedure.

In general, the technical steps are as follows:

1) The skin over the nose is separated from the cartilaginous vault. This is done as described by Jacqes Joseph of Berlin, for intranasal rhinoplasty. Intranasal incisions are made between the triangular and alar cartilages, first on one side, then the other. The two incisions are then united and the lower end of the septum is exposed by incising the membranous septum from its superior tip down to the spine of the superior maxillæ.

3) The triangular cartilages are split from their attachment to the septal cartilage.

4) The septal cartilage is mobilized superiorly.

5) The septal cartilage is then freed from any pathological attachment inferiorly.

6) A triangular piece of cartilage and nasal mucosa is excised from the larger of the triangular cartilages.

7) The septum is then sutured to this cartilage by means of a mattress suture of heavy silk.

8) The skin over the nose is allowed to readjust itself to the new framework of the nose.

The deformity must be overcorrected, since the gap left between the septum and the smaller triangular cartilage must be filled in with scar tissue which, in the process of healing, will tend to re-

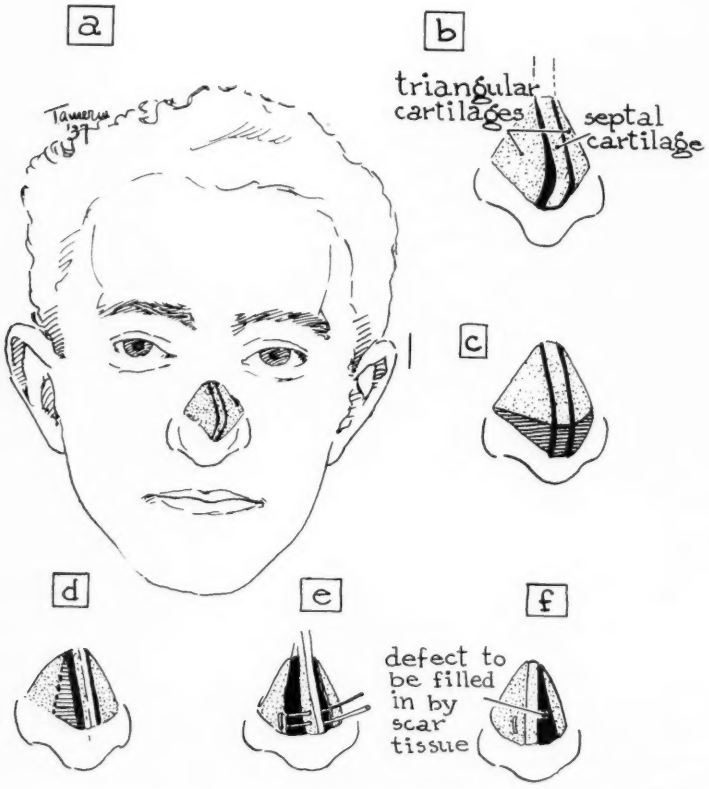

Fig. 2. Diagramatic representation of the operative correction in Case 1.

a—Diagramatic view of condition of septum and triangular cartilages in Fig. 2a. Note that the triangular cartilage on the side opposite the deviation of the septum is the larger of the two triangular cartilages.

b—Triangular cartilages separated from the septum.

c—Striped area indicates the amount of anterior end of the septum and triangular cartilages to be excised in shortening of the nose.

d—Striped area indicates the amount of triangular cartilage to be excised in equalizing the two triangular cartilages.

e—Mattress suture from the triangular cartilage through the septum.

f—Septum straightened and attached.

produce the deformity. It is this late contraction that may vitiate to some degree the initial result.

Modifications of this technique will depend upon whether there is an associated hump of the nose or elongation of the nasal tip.

If there is a nasal hump, its removal will free the septum from the triangular cartilages.

If there is an elongation of the nasal tip, the anterior end of the septum should first be shortened and a shortening of the triangular cartilages performed. Then the septum is attached to the triangular cartilages.

Other rhinoplastic procedures, such as an adjustment of the alar cartilages, the columella and alae nasae may be undertaken at the same time.

In the case illustrated, a submucous resection was performed prior to the nasal reconstruction. The necessity for the submucous resection depends upon the existence of posterior obstruction of the septum.

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XX

THE ROENTGENOGRAM AS AN AID IN THE DIAGNOSIS OF SURGICAL MASTOIDITIS

Comparison of Operative and Roentgen Findings in One Hundred Cases of Mastoiditis

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In this study, the operative and roentgen findings of one hundred consecutive cases of mastoid disease, which came to operation, were studied. The operative findings had been carefully described by the various surgeons and could be welded into this study, since all the surgeons were trained on the otologic service at the University of Michigan Hospital in Ann Arbor, and their reports followed a long established routine. The x-ray films were reviewed before reference to case histories and surgical findings was made, and in the interpretation of the films, there was constant consultation with the department of roentgenology of the University Hospital. The only element of selection of cases was the elimination of all children under five years of age, for the films of most of these had been taken by flash technique, which frequently does not produce results which permit the careful analysis carried out in this study.

By using the methods of study already outlined, it was believed that an accurate determination of the value of the roentgen ray in the diagnosis of mastoid disease could be reached and that the statistical errors of such points as the presence or absence of clouding of the mastoid, the presence or absence of coalescence of mastoid cells, its degree and location, and condition of the sigmoid plate and petrosa, could be estimated.

The roentgenograms include stereoscopic films of each side in three projections, full lateral, oblique postero-anterior, and full antero-posterior, using the Bucky diaphragm. The lateral view conforms closely to a projection described by Schüller. The head is

so placed that the sagittal plane is parallel to the film and the beam is directed through the top of the skull at an angle of thirty degrees, so that the direct ray passes through the external auditory meatus of the side examined. For the oblique postero-anterior, or Stenver's view, the head is placed so that the supraorbital ridge, nose and zygoma on the side to be examined are in contact with the plate and the beam is directed from below at an angle of twelve degrees and is centered on the mastoid process. For the antero-posterior view, the occiput is placed on the film and the beam directed in the sagittal plane along a plane passing through the hair line in front and the external auditory canals.

Occipitomental views to delineate the structures of the petrous pyramids and the base of the skull were not taken in any of these cases. Among the acute cases there was usually less than twenty-four hours between the taking of the films and the mastoidectomy. In the three cases in which the interval was seventy-two hours between roentgen examination and operation, there was no discrepancy between x-ray and operative findings. In five of the chronic cases, roentgen examination antedated the operation by one to seven or eight weeks, but again the operative findings paralleled the x-ray findings.

DURATION OF INFECTION AND TYPE OF OPERATION

Duration	No. of Cases	Complete	Radical	Petromastoid
Years				
9 or more	22	—	22	—
8	3	1	2	—
7	2	—	2	—
6	5	—	5	—
5	1	—	1	—
4	4	1	3	—
3	3	2	1	—
2	1	—	1	—
1	4	1	3	—
Months				
7	1	—	—	1
6	2	1	—	1
5	1	1	—	—
3	1	1	—	—
2	4	4	—	—
1	12	12	—	—
Weeks				
4	1	1	—	—
3	12	12	—	—
2	17	16	—	1
1	4	4	—	—

Sixty-six males and thirty-four females are included in the hundred cases. Fifty-four mastoidectomies were done on the right side and forty-six on the left. Among the hundred cases, forty-eight were classed as acute, and fifty-two as chronic, using three months' duration of otorrhea as the arbitrary dividing line between acute and chronic cases. Fifty-seven complete, forty radical, and three petromastoidectomies were done.

Sixteen of the one hundred cases had had a previous mastoidectomy. Among these sixteen cases, the operative defect was recognized by roentgenogram in fourteen, and two sets of films failed to show any evidence of previous surgery. In no case was an operative defect described in the roentgenogram which was not actually present.

From the roentgenograms it was attempted to determine whether the cellular development was limited, average, or extensive, and these results were compared with the actual operative findings. At operation, twenty-one mastoids were reported as small, the roentgenograms of which were interpreted as showing small mastoid development in fifteen, and average development in six. Forty-nine cases had medium or average sized mastoids at operation, of which fourteen were regarded as small, twenty-four as medium sized, and eleven as large by roentgen studies. There were thirty large mastoids found at operation, the roentgenograms of which were interpreted as showing small mastoids in three cases, medium in fourteen, and large mastoids in thirteen. Although in no instance of an actually small mastoid did the interpretation of the films show extensive development of the mastoid, in three cases of the thirty large mastoids, the roentgenogram led us to believe that a small mastoid would be found at operation.

SIZE OF MASTOID AT OPERATION AND BY ROENTGENOGRAM

Operation	X-ray		
Small 21	Small 15	Average 6	Large 0
Medium 49	Small 14	Average 24	Large 11
Large 30	Small 3	Average 14	Large 13

Diffuse clouding of the mastoid process in the roentgen study was present in all cases. Also, in no single instance was a normal mastoid found at operation. The single element of clouding of the cells has played no part in this study, for there is no single operative finding with which it can be correlated. It is generally recognized that more than mere clouding of the mastoid cells is needed to diagnose surgical mastoiditis.

Bone destruction, however, is a much more important point. At operation bone destruction was found in eighty-six of the one hundred cases. Among these, destruction was read in the roentgenograms in sixty-eight cases, and not seen in eighteen cases. Only once was destruction described in the roentgenogram among the fourteen cases which failed to show it at operation.

BONE DESTRUCTION AT OPERATION AND IN THE ROENTGENOGRAM

Operation	X-ray	
Present 86	Present 68	Absent 18
Absent 14	Present 1	Absent 13

A further attempt was made to localize the areas of destruction to the mastoid tip, peri-antral region, postsigmoid cells, sigmoid plate, petrous ridge and zygoma.

What was found to be minimal destruction of the tip at operation was similarly interpreted or not seen at all in the roentgenogram. Where there was moderate destruction of the tip, it failed to appear in the films in half the cases, and in the others it was described as minimal or moderate. Where there was extensive destruction it was not recognized at all in half the cases, and in the other half it was called minimal. Moderate destruction was described from the roentgen study of four cases where it was absent at operation.

TIP DESTRUCTION AT OPERATION AND IN THE ROENTGENOGRAM

Operation	X-ray			
Minimal 21	None 14	Minimal 6	Moderate 1	Extensive 0
Moderate 16	None 8	Minimal 3	Moderate 5	Extensive 0
Extensive 4	None 2	Minimal 2	Moderate 0	Extensive 0
Absent 59	None 45	Minimal 10	Moderate 4	Extensive 0

Although slight peri-antral destruction was found at operation in twenty-four cases, it was interpreted as absent in six cases, minimal in six, moderate in eight, and extensive in four, from the films. In the fifty cases where destruction was present, it was not recognized in twelve instances. We failed to recognize some degree of peri-antral destruction in only one case among the nine in which there was extensive destruction at operation. On the other hand, moderate or extensive destruction was described in six of the seventeen cases which showed none at operation.

PERI-ANTRAL DESTRUCTION AT OPERATION AND BY ROENTGENOGRAM

Operation	X-Ray			
Minimal 24	None 6	Minimal 6	Moderate 8	Extensive 4
Moderate 50	None 12	Minimal 8	Moderate 19	Extensive 11
Extensive 9	None 1	Minimal 0	Moderate 6	Extensive 2
Absent 17	None 3	Minimal 8	Moderate 5	Extensive 1

Of the seventeen cases where there was minimal postsigmoid destruction, there was complete failure to recognize it in the x-ray films in twelve cases. Likewise, we did not see any destruction in the postsigmoid area in eight cases among the fifteen found to have moderate destruction at operation. The one case with extensive postsigmoid destruction was interpreted from the film as showing no destruction in that area. Minimal and moderate destruction were described eighteen times among the sixty-seven cases which showed no actual destruction at operation.

POSTSIGMOID DESTRUCTION AT OPERATION AND BY ROENTGENOGRAM

Operation		X-Ray								
Minimal	17	None	12	Minimal	1	Moderate	3	Extensive	1
Moderate	15	None	8	Minimal	2	Moderate	4	Extensive	1
Extensive	1	None	1	Minimal	0	Moderate	0	Extensive	0
Absent	67	None	49	Minimal	11	Moderate	7	Extensive	0

The mastoid films did not show change in the sigmoid plate in ten cases which demonstrated moderate destruction of this area upon operation, nor did they show evidence in three similar cases which disclosed extensive necrosis at the time of surgical exposure. Among the twenty-one cases with minimal involvement of the plate, a change was recognized in the roentgenogram in only four cases. There were twelve perisinus abscesses and disintegration of the plate was recognized only once by x-ray.

SIGMOID PLATE DESTRUCTION AT OPERATION AND BY ROENTGENOGRAM

Operation		X-Ray								
Minimal	21	None	17	Minimal	3	Moderate	1	Extensive	0
Moderate	10	None	10	Minimal	0	Moderate	0	Extensive	0
Extensive	3	None	3	Minimal	0	Moderate	0	Extensive	0
Absent	66	None	64	Minimal	2	Moderate	0	Extensive	0

Five cases showed definite necrosis of the petrosa, but in no case did the roentgenograms point the way to this change. Conversely, petrous pyramid destruction was not described when it was not present. In the films of these one hundred cases there were no changes suggesting necrosis of the petrosa.

DESTRUCTION OF THE PETROSA AT OPERATION AND BY ROENTGENOGRAM

Operation		X-ray	
Minimal	3	None	3
Moderate	1	None	1
Extensive	1	None	1
Absent	95	None	95

We recognized destruction of the zygoma only once among the thirty-one cases with minimal and moderate necrosis. As in the petrous pyramid, we did not falsely describe destruction at any time.

DESTRUCTION OF THE ZYGOMA AT OPERATION AND BY ROENTGENOGRAM

Operation		X-ray			
Minimal	27	None	26	Minimal	1
Moderate	4	None	4	Minimal	0
Extensive	0	None	0	Minimal	0
Absent	69	None	69	Minimal	0

Cholesteatoma was found in eleven cases, but was recognized in the roentgen study only four times. One cholesteatoma was described in reports from the films and was not found at operation.

Twenty-four subperiosteal abscesses and four eroded semicircular canals were found at operation without any indication of their presence in the roentgenograms.

Analysis of these data, we believe, should aid in placing roentgen films in the diagnosis of surgical mastoiditis in a less equivocal position. It is generally agreed that mere clouding of the mastoid cells is not indicative of surgical mastoid disease. The films are of value in anticipating the size of the mastoid upon which one is to operate. We have found that bone necrosis can be recognized in the roentgenogram in about 80 per cent of the cases, but to attempt to localize accurately such destruction is impossible. Our efforts at roentgen recognition of disease of the sigmoid plate, semicircular canals, and petrosa were wholly unsatisfactory. Gross formation of cholesteatoma was recognized in only four of eleven cases. We believe that the otologist should depend upon careful and repeated clinical examinations for the greatest aid in establishing the diagnosis of surgical mastoiditis.

CONCLUSIONS

1. The clinical examination is of prime importance.

2. The roentgenogram is never entirely negative when a mastoid disease is found surgically, and probably is never clouded when the mastoid process is found to be normal at operation.

3. X-rays aid in demonstrating the general architecture of the mastoid.

4. The roentgenogram is 80 per cent efficient in indicating the presence of bone destruction.

5. The x-ray film is of little value in accurately localizing bone destruction or in determining its extent.

6. Erosion of the mastoid cortex, erosion of the semicircular canals, and necrosis of the petrosa were not seen in the films reviewed, though found at operation in several instances.

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Clinical Notes

XXI

INNER EAR DEAFNESS FROM TETANUS ANTITOXIN INJECTION*

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NEW HAVEN

Neurological complications following the injection of tetanus antitoxin either prophylactically or for the treatment of tetanus, are exceedingly rare. While serious and of definite medico-legal importance, they are in no way a contraindication to the judicious therapeutic or prophylactic use of tetanus antitoxin. However, their very rareness makes it important to report them, and for this reason the following case should be of interest.

REPORT OF A CASE

CASE 1.—The patient, a white boy, aged 15 years, was bitten on the right index finger by a white rat while assisting in an experiment at school on November 17, 1936. The wound was treated immediately and healed normally within a few days, according to the mother. That night the patient was given 3,000 units of tetanus antitoxin intramuscularly in the right arm. There was no immediate reaction. Nine or ten days later the boy had an attack of illness characterized by swelling of the right arm, fever (?) (his mother said he looked feverish but did not take his temperature), and malaise which lasted for five days. The patient was very dizzy at this time. There was a marked tendency to fall, and he had to be helped to the bathroom. Objects seemed to whirl around, though the patient does not remember in what direction. The patient was not seen by a physician during this period. Five days after the onset of the local reaction, at the time that the swelling of the arm had almost subsided, the dizziness had disappeared, and the patient definitely felt better, he awoke one morning (December 1) and noticed that he was very deaf. He did not have and never has had subsequently any noises in the ears. His mother noticed that ordinary speech, or even loud talking, was not heard by the patient. She

*Presented before the New England Otological Society, November 16, 1937.

has had to almost shout to make him hear. She has noticed no improvement in his hearing since then. His general health has gradually improved, and the boy has been back in school since January, 1937. He cannot keep up in his work, however, and is being sent to a class for lip reading.

PAST HISTORY

The patient has always been very healthy. He has never been treated by a physician except for measles and chickenpox. He has had very few colds and these have never been complicated by ear or sinus trouble. According to the patient, his mother, and the school authorities, his hearing was normal until the present illness. At the age of five he was Schick tested and presumably had toxin antitoxin for the prevention of diphtheria. He has had no operations and no severe accidents. He has never had tetanus antitoxin previous to November 17, 1936. He has never had infantile eczema, hay fever, asthma, urticaria or any food idiosyncrasy. There is no history of any hereditary disease—no history of epilepsy or any disease of the central nervous system.

EXAMINATION

The physical examination was made by Dr. George Blumer and showed no abnormalities whatsoever, except a slight generalized enlargement of the lymph nodes. A Wassermann reaction was negative. The examination of the head (exclusive of the ears) showed no abnormalities except enlargement of the tonsils and adenoids, which, according to the history, have never caused any trouble.

Ear drums.—First examination April 24, 1937 (five months after deafness began). Both ear drums are intact and appear essentially normal. The left drum is slightly more retracted than the right, but not enough to be called pathological.

Hearing.—The patient does not hear ordinary conversation in a small room at a distance of five feet when the room is darkened (as when transilluminating the sinuses). He does not hear whispers under any circumstances. He can hear loud conversation at a distance of five feet (normal about thirty-five feet).

Tuning forks—air conduction.—Forks C64 to C1024 are heard poorly in both ears. They are heard somewhat better on the right than on the left. C2048 is not heard at all on the left and heard very faintly on the right. The Galton whistle, while not an accurate test, cannot be heard on either side while using its maximum intensity. Air conduction is better than bone conduction on both sides.

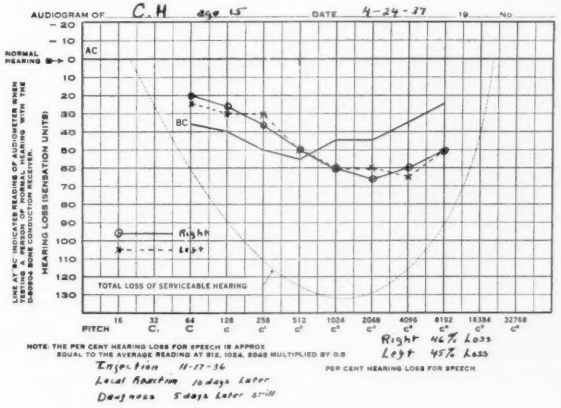
AUDIOGRAM OF C.H age 15 DATE 4-24-37 19 No
NORMAL HEARING
AC
BC
Right
Left
TOTAL LOSS OF SERVICEABLE HEARING
HEARING LOSS (SENSATION UNITS)
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C1 C c c1 c2 c3 c4 c5 c6 c7
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX. EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8
PER CENT HEARING LOSS FOR SPEECH
Right 46% Loss
Left 45% Loss
Injection 11-17-36
Local Reaction 10 days Later
Deafness 5 days Later still

Fig. 1

Bone conduction, using an AI fork, is heard seven seconds on each side, as contrasted with the normal of twenty seconds for me. The Weber test is not referred.

Audiometer test.—Shows a gradual loss of hearing for the higher tones. The hearing in both ears follows essentially the same curve. The right shows a loss of 46 per cent and the left 45 per cent (see Fig. 1).

Vestibular tests—Caloric test.—No nystagmus is produced by irrigating with a quart of tap water on each side with the patient in the upright position and also bent forward 90 degrees. There are no subjective sensations of dizziness. A Romberg test made immediately afterwards is essentially negative.

Turning tests—patient upright. Turning ten times to the right produces a horizontal and rotary nystagmus to the left lasting about 35 seconds. Turning ten times to the left produces a horizontal and rotary nystagmus to the right lasting about 40 seconds. The patient has definite subjective symptoms of dizziness but no nausea following the turning.

Inflation.—Both eustachian tubes were readily inflated and were found to be open. Inflation was done using a nasopharyngoscope. No pathological changes were noted besides the enlarged adenoids, which in no way interfered with the eustachian orifices.

SUBSEQUENT EXAMINATIONS

During the next seven months the patient was examined five times. Audiometer tests were done on each occasion. They never varied more than 5 per cent to 10 per cent from the original audiogram. The audiometer tests were not made in a sound-proof room, so that there may be errors of 10 per cent from normal.

Every effort was made to rule out the possibility of hysterical deafness, and on no occasion was any deviation from the original examination found.

On the second visit the caloric test was repeated, using a quart of ice water instead of tap water on each side, but no reaction was obtained. On a subsequent test ice water was directed directly onto the drum through a long trocar. It was necessary to use 30 cc. of ice water before any nystagmus was produced and even this was questionable.

DIAGNOSIS

The repeated examinations led to the diagnosis of inner ear, or perception, deafness. Hysterical deafness can definitely be excluded, and principally on the basis of the repeated audiograms, all of which do not vary by more than 10 per cent from the original pattern. The lack of response to the caloric tests while there was a normal response to the turning test, indicates partial damage to the vestibular tract which is permanent but quiescent.

SUMMARY

In summary, a normal healthy boy of 15 was bitten on the finger by an experimental white rat and that same evening was given a prophylactic dose of tetanus antitoxin. The rat bite wound healed without any difficulty and plays no röle in the subsequent events. Ten days after the inoculation the boy developed a general reaction, accompanied by a local swelling at the site of the inoculation. After being sick with this reaction for four or five days, including marked symptoms of dizziness, he suddenly developed bilateral deafness which has persisted since then. Physical examination, aside from the ears, showed no gross abnormalities.

COMMENT

The attack of sickness which occurred ten days after the injury apparently should be ascribed to a serum reaction, although some of the manifestations which are ordinarily seen, such as urticaria and joint pains, were absent. There was a definite history of a previous

inoculation in childhood. There was a definite local reaction at the point of the inoculation and a general reaction. It has been suggested that the reaction involving both eighth nerves was an anaphylactic reaction and very similar to an attack of angioneurotic edema. The patient undoubtedly had a diffuse serous labyrinthitis with a resulting partial destruction of function of both parts of the labyrinth. This was a permanent effect, which was evidenced by a hyposensitivity of the semi-circular canals as shown by their loss of reaction to the caloric test (sensitive) with a normal response to the turning test (violent). It was also shown by the loss of hearing of approximately 45 per cent on both sides. Both of these changes have persisted for a year after the injury without any appreciable change.

Experimentally, similar anaphylactic changes have been produced in animals by Lurie[1] with an audiometer curve very similar to the one noticed in this patient. It differs somewhat from the usual diagram of nerve deafness, in that there is a rather gradual loss of the higher tones, with a slight rise for the highest tone rather than a more sudden drop for these very high notes. Also Crowe[2] has seen a case of total deafness due, he thought, to angioneurotic edema, since the hearing returned within a short time after giving adrenalin.

Doyle[3] in 1933 summarized 49 cases of neurological complications of serum sickness reported in the literature, 34 of which followed the use of tetanus antitoxin. The majority of these complications involved the brachial plexus, though a few involved the optic nerve. The average interval between the onset of serum sickness and the neurological complication was two days. Some of the cases recovered and some did not. Doyle does not mention a single case involving the eighth nerve. Amberg and Hewitt[4] in 1935 reported a case of inner ear deafness in a boy aged 17 who had been given 120,000 units of tetanus antitoxin for the treatment of tetanus. The boy appeared totally deaf and this deafness did not clear up. During the treatment of the tetanus the boy had been given large doses of the barbiturates, so that the authors would not state the etiology of the deafness, ascribing it either to the tetanus, the tetanus antitoxin or the barbiturates. In 1936 Cutter[5] reported a case of inner ear deafness occurring in a boy of 14 who was given 125,000 units of tetanus antitoxin for the treatment of tetanus. The boy's deafness began one week after the administration of the tetanus antitoxin. It came on fairly gradually and was associated with noises in the ears and double vision. The audiometer test showed 69 per cent loss of hearing on one side and 61 per cent on the other. During the next two months the boy improved tremendously, and the hearing

loss had dropped down to 21 per cent on one side and 9 per cent on the other. This author felt there was no question but that the nerve deafness resulted from the administration of the tetanus antitoxin.

In our case there is a definite perception type of deafness approximately 45 per cent on both sides, associated with an impairment of function of the vestibular tract. The patient has shown no improvement for one year since the onset of his deafness, so that it undoubtedly is permanent. Dizziness was present only during the initial acute attack. From the sequence of events and the nature of the lesion, there is no other conclusion which can be drawn but that the deafness is the direct result of the injection of the tetanus antitoxin.

It was felt important to report this case, as it followed the prophylactic use of tetanus antitoxin, while the two previously reported cases followed the use of large doses of antitetanus serum for the treatment of tetanus.

442 Temple St.

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XXII

CEREBROSPINAL RHINORRHEA WITH AUTOPSY REPORT*

JOHN J. SHEA, M.D.

MEMPHIS

Cerebrospinal rhinorrhea is not uncommon following fractures of the base of the skull. Many die of meningitis, but some cease to drain and when serious complications do not follow, their cases are not recorded. The frequency of this important complication cannot be accurately estimated and medical literature does not possess an accepted thesis on the subject, for only in a large neurosurgical service will any single operator encounter a sufficient number of spontaneous cases to be in position to speak authoritatively upon the subject.

Charles Miller,[1] in 1826, reported before the Edinburgh Medico-Chirurical Society the first of our cases in English, and King[2] recorded the second case in 1834. Little attention was paid to this subject until Sir St. Clair Thomson[3] brought it to the attention of the medical profession in 1899. Johnston,[4] in 1926, emphasized the value of the knowledge obtained at autopsy from reviewing the cases reported in the literature.

The three cases that I desire to report are of interest to otolaryngologists because the first followed a pituitary tumor operation (Cushing) in a patient upon whom I had previously performed a sphenoid operation. The other two are reported through the courtesy of Dr. R. Eustace Semmes. The second is of interest because the only nasal pathology present was some innocent-appearing polyps under the right middle turbinate, yet at autopsy these polyps were found to be the result of leakage of cerebrospinal fluid through the right ethmoid mass. The third was a patient with acromegaly, in whom the pituitary gland eroded into the sphenoid sinus. This was followed by the escape of fluid into the nose.

THE MECHANISM OF SPONTANEOUS RHINORRHEA

The cerebrospinal fluid is separated from the nasal sinuses and nares from within out by the arachnoid, the dura, a bony partition

*Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Georgia, January 24, 1938.

and the mucous membrane of the sinuses or nose. Tumors, inflammation or pressure may cause atrophy of the dura and the bone, after which any increase in pressure will rupture through, with escape of fluid.

Without previous disease in these barriers, traumatism may produce a fracture, with a tear of the dura, and allow the escape of cerebrospinal fluid. Once there is a free communication between the fluid spaces and the nasal cavities or sinuses, the ingress of infected material (fluid or air) depends upon the relative pressure between the two cavities rather than upon their hydrodynamics. As long as the fluid continues to pass outward, infection is unlikely; however, the intracranial pressure, which is negative in the erect position, becomes even lower with the escape of cerebrospinal fluid, so that if the opening is large and direct enough, air from the nose or sinuses, together with the secretions, is sucked back into the intracranial cavity or actually forced into it when the patient blows his nose, coughs or sneezes.

The risk of intracranial complications is greater when the tear is into the nose rather than into the sinuses, especially if the latter are in a healthy condition. The nature of the infection depends upon the nasal flora at the time the fistula develops. Meningitis may not develop until the patient contracts a cold.

The dura in childhood is loosely attached to the cranium and difficult to tear with fractures of the skull. In the adult it adheres closely and tears readily with fractures. The structure at the cribriform plate consists of thin bone with a firmly attached thin dura, and, for this reason, fractures here are dangerous and impossible of repair.

REPORT OF CASES

CASE 1.—S. F. B., age 38, first seen on February 3, 1928, with the history that one year before, after an acute rhinitis, the vision in the left eye failed suddenly and that a study of his nasal sinuses was reported negative. Recently a checkup of his sinuses revealed in the roentgenogram a cloudy left sphenoid and an enlargement of the sella turcica, with loss of the posterior clinoid processes. The eye findings (Dr. James B. Stanford) were vision right 20/15, left 20/200; the left disk was pale with an absolute scotoma in the inferior nasal quadrant. Our examination confirmed the possibility of the left sphenoid being the cause of the optic neuritis and, being supported by the loss of vision following an acute rhinitis and the site of the scotoma being in the inferior nasal quadrant, the

sphenoids were drained on May 9th. No improvement followed the operation, and on the 25th the patient was referred to Dr. Harvey Cushing of Boston for surgery of his pituitary gland. One year later he consulted the late Dr. R. C. Lynch of New Orleans, who advised radium therapy, which was accepted. Shortly after his return home, the patient became conscious of an intermittent flooding of his nose by a thin watery discharge. Each time that the patient reported after this he complained of intermittent discharge of the fluid, which undoubtedly was cerebrospinal fluid. Five months prior to death he consulted Dr. Walter E. Dandy of Baltimore, Maryland, who advised deep x-ray therapy.

Present History.—The patient had been feeling unusually well until the day before his admission into the hospital. He became unconscious after seven hours of headache and was brought from his home to Memphis for hospitalization under the care of Dr. R. Eustace Semmes.

Examination.—Middle aged male, obviously very ill, unconscious, moaning and very restless. Head, normal; neck, very rigid; pupils equal and reacted fairly well to light; optic disks were pale over the nasal half, extending on to the retina; the remaining cranial nerves were normal, as far as examination could elicit; the deep reflexes were hyper-active. There was a positive Babinski and a positive Kernig test. Lumbar puncture produced a turbid fluid under 250 mm. of pressure; 10 cc. of the fluid was removed. The smear showed many pus cells and gram positive diplococci, which, on culture, proved to be pneumococcus, type II. The patient lived less than twelve hours after his admission to the hospital.

Autopsy Report.—December 12, 1935, Laboratory No. 44103. "Gross findings on brain and cranial cavity: The subarachnoid space over the cortex contained surprisingly little purulent fluid. The cortex itself was somewhat congested and the convolutions were flattened, indicating the presence of increased intracranial pressure. The lateral ventricle contained a thick greenish-yellow pus. There was also a small amount of this material in the third and fourth ventricles.

"The rest of the significant findings were confined to the region of the sella turcica.

"When the frontal lobes were elevated the optic nerves were found to be normal. There was no evidence of flattening or pressure atrophy. The sella turcica was markedly enlarged. The anterior clinoids showed an erosion and the posterior clinoids had

completely disappeared. The sella turcica measured 4 cm. across in the antero-posterior dimension and 3 cm. in the lateral. It was filled by a large tumor which had numerous hemorrhagic areas throughout. In fact, the whole central part of the tumor seemed to be necrotic. The tumor itself extended laterally on the right side to the foramina rotundum and ovale and had eroded away a large area of the greater wing of the sphenoid at this point. On the left the tumor probably extended 1 cm. laterally. As previously noted, the posterior clinoid processes had been completely destroyed, likewise the dorsum sella had been considerably eroded and the tumor extended over this prominence through the incisura to a point about half way between the dorsum sella and the foramen magnum.

"After the tumor had been removed the floor of the sella turcica could be examined. There was a defect in the floor about ½-cm. in diameter through which the tumor had eroded. This opened directly into the sphenoid sinus. Examination of the sphenoid sinus failed to reveal any tumor tissue whatever. However, the lining of the sphenoid was markedly swollen and was covered with a thin film of yellow purulent material.

"Diagnosis: Pituitary tumor—chromophobe adenoma."

CASE 2.—W. R. B., age 26, was first seen in the autopsy room.

Past History.—Thirteen years ago, Dr. W. W. Adson of the Mayo Clinic performed a suboccipital craniotomy, but failed to locate a tumor. This was evidently a basal arachnoiditis causing the obstructive hydrocephalus. The lad recovered from the operation and received an extensive course of radium treatments and his symptoms subsided. The relief was due undoubtedly to the decompression and drainage. Several weeks after the operation a thin watery fluid began to drain intermittently from the right side of the nose. The interval between the attacks lengthened until it occurred only once or twice a year, but recently the flow has been continuous. Immediately after the drainage he would have a headache above the eyes. An examination of this fluid was as follows: Protein, faint trace; sugar, 40 mgm., /100 cc.; chlorides (as NaCl) 700 mgm., /100 cc.

The following is an abstract from Dr. W. W. Adson's letter to Dr. B. S. Guyton of Oxford, Mississippi: "The patient returned to the Clinic in 1927 for re-examination. He still presented evidence of intracranial pressure, but since I had been unable to demonstrate a tumor in the posterior fossa during the previous operation, I did not feel that I could advise further surgery without any more

Fig. 1. Case 2. Illustrating erosion through the roof of the right ethmoid and beginning depression into left ethmoid roof.

localizing supra-tentorial symptoms. His ophthalmologic examination made on July 19, 1927, showed some atrophy of both optic nerve disks and an increased blind spot with a vision of 6/10 in the left with corrections and 6/7 in the right with corrections. There was no choked disk. There has been some correspondence from time to time but no further surgery or x-ray treatment has been advised or carried out.

"I am unable to explain any relationship between the operation and his present difficulty because the operation was carried out in the back part of his head. My guess would be that he has a draining third ventricle which has probably eroded through into the

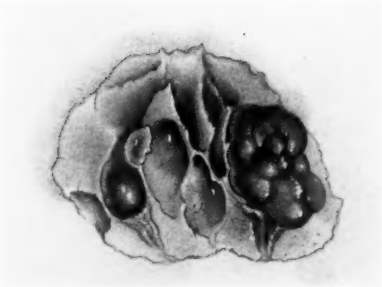

Fig. 2. Case 2. Illustrating polyps of right ethmoid cells.

sphenoid and when the pressure increases the fluid undoubtedly finds its way into the sphenoid and then into the nasopharynx."

"Pathological note.—At autopsy the erosion was found through the right ethmoid."

Present History.—One week ago he awoke with nausea, vomiting and a severe headache, at which time he was seen by Dr. B. S. Guyton. The patient was semi-conscious, but cleared up the following day. The lad was in the University of Mississippi Infirmary, and, while going to the bathroom, fell down the stairs, striking his head, but did not lose consciousness. Yesterday all the symptoms increased and the patient became unconscious and remained so until death.

The lad was transferred to the Baptist Memorial Hospital of Memphis, under the care of Dr. R. Eustace Semmes.

Examination.—Patient unconscious; right pupil dilated; disk of both eyes blurred; lower half of right facial nerve showed a distinct weakness; there was a left hemiplegia with increased reflexes on the left; Babinski test was positive; the neck was slightly stiff; Kernig test was negative; the suboccipital decompression area was bulged and slightly tense; the spinal fluid was bloody.

Autopsy Report.—June 18, 1937, Lab. No. 50205. "Head, no external marks of violence. Internal examination: the brain is of normal weight, covered by a massive subdural hemorrhage on the right side extending from the mid-portion of the brain to the ex-

treme right base, pushing the entire brain substance to the left, invaginating the thin cortex into the greatly dilated ventricle and thus not producing pressure. This clot was composed of approximately 500 cc. of blood which came from a ruptured vein on the dorsolateral portion of the brain. A very marked hydrocephalus was present with a communication between the right anterior fossa and the nose through a fistula in the roof of the right ethmoidal capsule. The right anterior ethmoid cells were filled with hemorrhagic polyps."

Comments:

1. The symptoms for which the original brain operation was performed were those of the internal hydrocephalus.

2. The site of the fistula is unusual.

3. In reviewing this case, the patient evidently had an arachnoiditis causing an internal hydrocephalus, the pressure of which thinned the dura and the roof of the right ethmoid cells, producing a leak which led to the growth of the polyps. When meningitis developed he attempted, in his delirium to descend the steps, but fell and struck his head, not hard enough to fracture the skull or render him unconscious, but enough to cause the right hemisphere to sag away from the skull and to tear a vein in the subdural space. The accumulation of blood in this space resulted in a contralateral weakness and the involvement of the third cranial nerve with a dilation of the pupil. The usual evidence of a subdural hematoma is a marked increase of intracranial pressure, but in this instance it was absent. The explanation of this phenomenon was that the tremendously dilated ventricle was invaginated by the clot, displacing the intraventricular fluid into the nose.

CASE 3.—Mrs. E. J. R., age 45, consulted Dr. R. Eustace Semmes, giving as her chief complaints headache, loss of vision and enlargement of hands, feet and features. The neurological examination was typical of acromegaly with an optic atrophy with choked disks. Nasal examination was negative.

Roentgenological study revealed the following: October 31, 1929. "Skull shows definite erosion over the sella turcica which has destroyed both clinoids and fills a considerable portion of the sphenoid sinus. Some rarefaction of the inner tables, probably due to increased pressure. Impression: intracranial neoplasm. Enlargement of the cranial bones and jaw suggesting acromegaly." (Dr. W. A. Runkle.)

The diagnosis was made of pituitary tumor and the patient was given x-ray therapy, which was followed by relief of the headache, improvement of vision and an escape of serosanguineous fluid from the nose. She was under treatment for three years, during which interval she had a series of five x-ray treatments, the patient remaining active and fairly comfortable with useful vision. Three months prior to death she developed cerebrospinal rhinorrhea, which terminated in a septic meningitis.

RHINOLOGICAL CONSIDERATION

Whenever a patient gives a history of intermittent flooding of one naris by a thin watery discharge, the possibility of cerebrospinal rhinorrhea must be considered. If the diagnosis is made either in a traumatic or spontaneous case, the patient should be warned as to the risks, the greatest of which is the contraction of an acute cold; and should be impressed with the necessity of determining the cause of the rhinorrhea. On one occasion, twenty-one days after discharge from the hospital, a woman who had suffered a fracture into the left frontal sinus, was returned with an acute meningitis. Her history was that she contracted what appeared to be a simple rhinitis, but in two days she became unconscious and developed meningitis.

These patients should be warned against blowing their noses or sneezing and certainly should avoid nasal douches. The treatment of the nose during an acute flooding should be that of watchful waiting, rest in bed and isolation from any possible contact of infection.

SURGERY

Advances in intracranial surgery has made possible the repair of some of the traumatic fistulas. In one instance previously reported by Dr. Richmond McKinney, Dr. R. Eustace Semmes repaired a hole in the posterior wall of the frontal sinus by replacing the fragments of bone and patching the tear in the dura with periosteum. The patient made an uneventful recovery.

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XXIII

TUBERCULOSIS OF THE PETROUS PYRAMID*

LOUIS KLEINFELD, M.D.
AND
GEORGE Y. SMITH, M.D.

NEW YORK

A 3-year-old Porto Rican female infant was admitted in February, 1936, with the complaint of diarrhea and loss of weight. A year before admission the patient had had several attacks of earache and two months before admission a discharge from the right ear. On admission routine examination was negative except for a pulsating discharge from the right ear. Mantoux was negative. Examination of the stool disclosed the ova of *schistosoma mansoni*. The child had a fever with evening rises to 102. X-ray of the chest showed infiltration of the right hilus. The child was treated with tartar emetic intravenously and the ova disappeared from the stool.

A month after admission a right facial paralysis was noted and necrotic cheesy material was seen in the canal of the right ear. A large perforation of the drum was present. X-ray of the mastoid showed merely increased density. A radical mastoidectomy was done and a slight amount of destruction was noted in the antral region. Culture of the mastoid produced a growth of streptococcus viridans. Within two weeks the facial paralysis disappeared but the fever and diarrhea persisted. However, a month later, the right facial paralysis reappeared. The lower angle of the post-auricular wound was red, tender and swollen. Neurological examination of the facial nerve showed no response to faradic, and a slow response to galvanic stimulation, the impression being that there was present a reaction of degeneration of the right facial nerve. On May 12th, six weeks after the reappearance of the facial paralysis, marked fluctuation was noted over the mastoid scar. The external canal became considerably narrowed and there was profuse pulsating discharge. The post-auricular scar was reopened; smears and culture were negative for tuberculosis. Guinea pig inoculation resulted in the death of the animal in a few days. Five days later a revision of the right radical operation was done by the author. Necrotic bone was found in the

*From the service of Dr. L. E. Hetrick, Metropolitan Hospital, New York.

angle between the sinus and the middle fossa and also at the tip. The bone in these areas had a peculiar white "dead" appearance. A deep pit was found in the primary groove below the posterior semicircular canal. The dura, which had been exposed at the previous operation, was found adherent to the external auditory canal and covered with granulation tissue. The posterior canal wall could be traced down about half way, below which it was replaced by granulation tissue. A fibrous strand was seen passing through this area and was thought to be the facial nerve. The patient later developed a sub-perichondrial abscess, which was drained. Repeated smears, cultures and biopsies for tuberculosis were all negative. Another Mantoux test was done (1-100) and reported negative. Spinal fluid showed five lymphocytes, no increase in globulin, sugar 66 mgm. per 100 cc., no organisms on smear or culture. The postauricular wound then began to drain a grayish, caseous material. The child's condition became worse, and it finally died in October, 1936.

The points of interest at autopsy were:

1. Evidence of miliary tuberculosis and tuberculous cavitation of the chest in spite of a negative Mantoux test.

2. Evidence of an old infection of the intestines and liver by schistosoma.

3. On the anterior surface of the right petrous pyramid two fairly large elevations were seen, one of which communicated with a necrotic area, which had eroded into the facial canal. These elevations both proved to be tuberculomata.

SUMMARY

This child, with a combined infection of tuberculosis and schistosoma, developed a discharging ear requiring radical mastoidectomy. Although laboratory and clinical examinations during life failed to reveal tuberculosis, the appearance of the ear was so strongly suspicious that numerous smears, cultures and specimens were taken and no evidence of tuberculosis found. Guinea pig inoculation was unsuccessful because of the prompt death of the animal. The facial paralysis may have been due either to tuberculous involvement of the lower part of the posterior canal wall or to tuberculous involvement of the petrous portion of the facial nerve.

The persistence of discharge immediately following a fairly complete radical operation should have led to suspicion of petrositis.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 1, 1937

THE PRESIDENT, DR. WALTER H. THEOBALD, IN THE CHAIR

CASE REPORTS

Experiments on the Conduction of Sound in the Ear

H. G. KOBRAK (by invitation)

(This paper appears in full in this issue.)

DISCUSSION

DR. J. R. LINDSAY: The work which Dr. Kobrak has just presented to you demonstrates the way in which the intrinsic muscles of the ear function, and the effect which they exert on the conduction of sound waves to the inner ear.

The relationship between the muscle contraction and the hearing sensation has been definitely established by our observations on the human, in over thirty of whom we have been able to observe the stapedius reflex.

These preliminary observations have laid the foundation for further investigations along two entirely different lines.

First, the effect which these muscles have upon the hearing under various pathological conditions. As Dr. Kobrak has said, this has been neglected and our information up to the present has been largely theory. Investigation along this line is under way.

Second, the reflex response of these muscles to sound provides an objective measure of cochlear function in the animal, and opens up an avenue for experimentation on hearing.

The middle ear muscle reflex involves the auditory tract only as high as the olivary nucleus and is therefore not affected by the higher centers. It therefore provides an ideal means of studying such questions as auditory fatigue, and the effect of masking. The

reflex is a relatively simple method of testing an animal's hearing when compared with the specialized technique necessary to condition animals or with the complicated electrical equipment necessary to obtain the electrical responses. The factors of attention and fatigue of higher centers are eliminated. Concerning the electrical method it may be said that until more is known about the origin of the electrical response it should not be assumed that it is an index of hearing under all circumstances.

DR. ALFRED LEWY: I would like to ask Dr. Kobrak to tell us at what frequencies he gets the reaction, and how Dr. Lindsay determines at what level in the brain that reflex operates. Were there sections made? Does Dr. Kobrak know of any experiments on the ears of patients who had a gasserian ganglion resection?

DR. E. J. BLONDER: I would like to know how you localize the stimulation to the olivary nucleus. Are you able to cut the olivary nucleus or do you make a complete section? There are some pathways from the cochlear nuclei to the olivary nucleus, and I wonder whether your method possibly injures some of these tracts before the olivary nucleus is reached?

DR. T. C. GALLOWAY: It must be obvious that the tone is heard without the reflex being active, because the tone must precede the reflex and activate it. Is then the reflex essential to hearing?

DR. GEORGE DENNIS: Will you describe the pathway of the reflex?

DR. H. G. KOBRAK (closing): In reply to the question of Dr. Lewy, the curve of the muscle contractions resembles the threshold of hearing. In the rabbit the optimum of the muscle reflex has been found at about 2,000 d.v. Unfortunately we do not know the optimum of hearing in the rabbit. In the human the optimum of muscle contractions was determined at about 1,500 d.v. In pathologic cases in which we observed the stapedius through a perforation the resonance frequency may be changed. The influence of a pathologic resonance frequency on hearing has never been studied. Dr. Lewy also asked whether a patient with extirpated gasserian ganglion has been examined as to hearing. No. However, there has been an investigation on patients with facial paralysis. In these cases the branch to the stapedius muscle was thought to be paralyzed. The hearing was found impaired. The author of this study concluded that his findings were a proof for the accommodation theory, since the loss of the muscle diminishes hearing. However, these conclusions are not definite.

Replying to Dr. Blonder as to the influence of olivary nucleus on the reflex, the central part of the reflex has been studied by Hammerschlag. By cutting experiments it was established that the trigeminal and facial nuclei are connected with the primary acoustic nucleus. Sections above those nuclei leave the reflex intact. So far as I know the olivary nucleus has not been mentioned in the literature as an essential part of the reflex arch.

In reply to Dr. Galloway, the effect of the muscle reflex cannot be determined by threshold tests. Our typical audiometer curves represent stimuli which are below the threshold of the muscle contractions. There may be some influence of the muscles on the loudness of over-threshold stimuli.

Control of Delayed Tonsil Hemorrhage

WILLIAM A. SMILEY

(Abstract)

Most of the articles in the literature are concerned with control of hemorrhage at the time of operation or a few hours later, and many ingenious devices have been provided for the application of pressure, placing of sutures or ligatures, while the virtues of astringents, caustics and hemostatics have been extolled.

Secondary hemorrhage has been mentioned occasionally as a possibility, and a few cases have been reported of hemorrhage starting between the fourth and fourteenth day, one recurring for twenty days. These hemorrhages occur mostly in adults, are more frequent after local than after general anesthesia, and are usually unilateral. They occur in about 2 per cent of adult tonsillectomies, usually between the fourth and seventh day, and usually in the early morning hours. The amount of blood lost may be only a few cc., or the patient may vomit several hundred cc. of blood and stomach contents.

In treating these hemorrhages, the clot which is usually present in the tonsil fossa is removed and the bleeding point visualized. In a series of twenty-nine patients in the past five years, the bleeding has been controlled by the superficial injection of 1 per cent novocain around the bleeding point. The amount of novocain used has never exceeded 1.5 cc. The action is explained thus: The fascia of the tonsil bed is slightly elevated by the injection, causing the bleeding vessel to be retracted below the surface. The bleeding stops promptly. In three instances, normal saline solution was substituted with satisfactory results, but one of the patients complained of considerable pain immediately following the injection.

Five patients in this group had failed to respond to ordinary methods of treatment, such as clot removal, ice pellets, ice bag to the throat, astringents or even caustics, and the bleeding had continued or recurred for over two hours. In no patient was there a return of bleeding. There was no resulting infections, and the throat was less sore than after the use of astringents or caustics.

DR. FRANCIS L. LEDERER: We cannot disagree with Dr. Smiley —he has positive evidence of twenty-nine cases. As to injecting or introducing a needle—where is he going to introduce it? He is going through an area of infection. I wonder if there is any thought in his mind of producing a parapharyngeal abscess as a result of injecting novocain or any other solution into these tissues. Not that the selution is not sterile, but through the introduction of organisms from the outside. That might be borne in mind in considering this method as a panacea. It is hard to visualize the mechanism, namely, edema producing retraction of or pressure on a vessel, although I have no better explanation for its action. There is adrenalin present in the solution. Perhaps that accounts for the momentary hemostasis. Were there any recurrences of bleeding?

DR. AUSTIN A. HAYDEN: I remember very well when Dr. Beck first brought out hemorrhage control in tonsillectomy by suturing a piece of gauze into the tonsil fossa, I think about 1912, perhaps a little earlier. The bleeding point should always be looked for and will usually be found. It seems to me that this is a primary consideration in any event, if hemorrhage is to be controlled by injection, suture, or what not. I agree with Dr. Smiley that pressure can stop that, and that the injection of any fluid, normal saline or antiseptic, can conceivably stop a hemorrhage, although I have never done it that way. I always put a suture in around the bleeding point or bleeding area. I would be very much afraid of doing what he has suggested because I think it would mean another call later on in the night, at four or five o'clock, and I am opposed to those calls.

DR. JOSEPH C. BECK: In discussing Dr. Smiley's paper, I wish to say that the number of secondaray hemorrhages in tonsillectomy that he reports is startling to me. I have removed a great many tonsils in my day, but I do not believe I can record three genuine cases of secondary bleeding; that is, if I understand Dr. Smiley's differentiation of a septic condition of the throat and bleeding that cannot be stopped because the blood vessels cannot retract. This method seems perfectly feasible if Dr. Smiley can avoid what Dr. Lederer has spoken of, namely, injecting infective material into a sterile field. However, I can readily see that injecting a large quantity of liquid

into the tissues about the bleeding point can cause a compression which may be sufficient to get a reaction that might close it. If there be a great deal of elevation, so that the loose tissue would contain the vessel below, it seems to me that a hematoma might develop. Suturing a septic field, attempting to tie a vessel, has not been very successful in my hands as it tears out. I think by using Dr. Smiley's method, which is very simple, we may get some good results.

DR. PALMER GOOD: I have been unfortunate in having had about ten secondary hemorrhages even though I have not done a lot of tonsillectomies. They have all seemed to me to be venous bleeding from the paratonsillar vein which runs vertically in the tonsil fossa. In order to have a secondary hemorrhage I believe that the vein must be left intact at the time of the operation, and that the wall of the vein sloughs through on the sixth or seventh day. Often the bleeding is of short duration and stops if the patient sits up and holds a piece of ice in his mouth. In one patient it was thought best to suture the bleeding point under a general anesthetic. The easiest and surest way that I have used is the double armed sponge holder so made that gentle pressure is exerted upon and between the tonsil fossæ. The patient is usually prepared with one-fourth grain of morphine. I am very much interested, however, in Dr. Smiley's method and will try it if I am unfortunate again.

DR. SAMUEL SALINGER: I would like to know on whose authority it was determined that a tonsil bed was a septic field. When the tonsils are removed you have a clean wound covered with a fibrinous exudate, and it never has appeared to me to be very septic. It is true that a large number of saprophitic bacteria may find their abode in that membrane, but they are not dangerous. If the field were septic then every time a man put a suture into the tissues he would have infection; certainly there would be more than have been reported. I say this to support Dr. Smiley's statement, that the mere introduction of a needle into the average tonsil bed is not likely to carry infection.

DR. L. B. BERNHEIMER: I recall an instance when infection did play an important role in the production of secondary tonsillar hemorrhage. This individual was a young man of about 18 years who bled profusely eighteen hours after a tonsillectomy. Examination revealed a profuse oozing from the right tonsillar fossa with a dissecting hematoma extending down the fascial planes of the neck over the carotid sheath. A decision was immediately made to ligate the external carotid. In exposing the vessel for ligation actual free blood and blood clots were encountered burrowing down between the superficial and the investing layers of the deep cervical fascias. Following liga-

tion of the external carotid, hemorrhage was controlled for about seventy-two hours, at the end of which time it recurred. The boy at this time showed clinical evidences of pharyngomaxillary infection, and was almost exsanguinated. The neck wound was reopened, exposing a pharyngomaxillary abscess which had eroded the internal carotid. The common carotid was then ligated and following repeated transfusions the patient recovered.

Dr. Robert Good: I think it would be a mistake to consider every case of secondary hemorrhage an infection. We can very often see the tonsil cavity on the sixth or eighth day containing remnants of dry membrane, and when this falls off it may open a blood vessel and produce hemorrhage. The only infections I have seen are where a blood clot is allowed to remain for a number of days in the tonsil cavity.

Dr. William A. Smiley (closing): I have not encountered any cases of general oozing from the tonsil fossa where I was not able to visualize the point from which the hemorrhage was originating.

With regard to Dr. Lederers' question, the point of injection has been a very small distance from the bleeding point, maybe 0.5 cm. and the needle has been introduced only about .25 cm. The amount of fluid injected has not, as I mentioned, been more than 1.5 cc. in any patient. The first time I used it, was in preparation for a suture. When I put in the anesthetic the bleeding stopped, and I waited, but it did not start to bleed again. I do not feel there is any more danger in introducing a hypodermic needle into the tonsil fossa than in introducing a needle with a suture attached. I have not used any clamp, either double inside clamp or inside-outside clamp, because I figured the discomfort to the patient was such that I preferred to avoid it if possible. Myerson's article in the American Journal of Surgery, early this year attributes little importance to infection in the tonsil fossa as an etiologic factor in these delayed hemorrhages. With regard to Dr. Bernheimer's case—I do not think I would be interested in trying novocain in such a case.

The Postoperative Repair of the Paranasal Sinuses: Experimental Studies and Their Practical Application

M. A. Glatt, M.D.

(Author's Abstract)

The author stated that the finding at operation on a maxillary sinus of bared walls and an atrophic state of the bone had stimulated him to study the postoperative repair of the sinuses.

He described his technic of surgical operation on the maxillary sinus without the establishment of a counter-opening into the nose. In other experiments the usual Caldwell-Luc technique was carried out, with and without the laying of a mucoperiosteal flap into the sinus.

The failure to establish a counter opening in the nose did not interfere with the satisfactory end results. In fact, the postoperative gross appearance of the sinus cavities, when examined by direct vision or with the nasopharyngoscope, were more favorable in those instances where no counter opening was made into the nose. The microscopic sections also showed less cellular infiltration. The cavities were much reduced in size and presented pale surfaces free from discharge.

In summarizing the reports of various investigators, he stated that their disagreement is due to either the interpretation of the end results of exceptional cases, or to too broad or too narrow a use of the term "regeneration." In comparing the results of animal experiments with those in the human, one must consider the difference in reaction to trauma of a normal and of a diseased tissue.

There is in the postoperative repair in the human a variation in the degree of obliteration of the sinus cavity by new bone and fibrous tissue due to local differences in bone regeneration occasioned by the infection. The surface becomes covered with epithelium which proliferates through the ostium or the counter opening. The character of the epithelium varies considerably; practically it does not matter what type covers it.

Invaginations of the surface epithelium have been noted more in those cases where a counter opening has been made in the nose. The structure of those invaginations is similar to that of the surface epithelium, with what appears to be glands. However, they cannot be considered as true glands. The author believes that these pseudoglands may lead to the formation of postoperative cysts.

Remnants of mucous membrane, which are accidentally or purposely left in the sinuses with the hope that they will unite with the newly formed epithelium or help to epithelize the sinus cavity, do not function well. They may become completely covered over or be embodied in the scar tissue causing no harm, or they may be a source of cyst formation and reinfection. Surgical procedures which consist of incomplete removal of the mucosa, are therefore, apt to be unsuccessful.

From the histologic studies one may deduct that after the complete removal of the mucoperiosteum in a case of uncomplicated

sinus disease, drainage, ventilation and epithelization of the area are of little importance in the attainment of satisfactory end results.

DISCUSSION

DR. JOSEPH C. BECK: Dr. Glatt allowed me to study the preparations you have seen on the screen and also to read his paper. Of course he did not have time to read a very important part of it in the review of the literature, which would have helped a great deal in understanding many points made. I would like to carry you back a little in the history of sinus disease. In the study of sinuses in Vienna and Berlin, by Hajeck and Jansen, and many men in this country, they all believed the lining of the sinuses should never be removed. That was my teaching for many years; in fact, until a short time ago. I have always been skeptical about the formation of a membrane completely leaving the bone, especially in a cavity the size of the antrum. In the subsequent reopening of an antrum which had been previously operated upon and in which the entire membrane had been removed, I have found it completely covered by a new membrane—I do not say healthy mucous membrane, or even mucous membrane. Most of it has the appearance of a membrane that is moist; the greater portion of it is smooth, and undoubtedly it functions approximately the same as the original membrane. I think there is a difference between the frontal sinus and the antrum and do not believe they can be compared to the lining of the bony cavity of the ear after radical mastoid, as here we have an entirely different epithelium. We know the epithelium in the ear reforms as a scar, and it is poorly nourished; there is constant desquamation going on.

Jansen has shown that after he experimentally skin grafted an antrum, there was constant desquamation within the cavity. As far as the sinus is concerned this does not take place either in the frontal or antrum if it is allowed to heal naturally as I have seen both of these after the membrane has been entirely removed. I have not seen true glands in the preparations shown here tonight or in others that I have studied. I have always felt that there were glands found in the region of the ostia in these reformed membranes, but they are pseudo-glands which may secrete and are in the form of folds. A number of years ago there was a report in the German literature by Tondorf who had the courage to operate upon the antrum and remove the entire membrane, using the greatest of care not to enter the nose in any way. He did not remove any of the bone; merely the entire lining of the antrum. After packing the cavity with gauze for forty-eight hours he subsequently reopened the antrum and removed some of the reformed membrane, which he examined

histologically. He saw the same type of membrane that we find in the relining of the antra. In some cases, after he had removed all of the membrane, he took small pieces of the underlying bone and sectioned them for microscopic examination. He found tissue in the crevices of the bone, which he could not identify as epithelium, but as epithelioid. He felt that these small nests might be the cause of this regeneration of the membrane. I have not seen any confirmatory work along that line. This work, as shown by Dr. Glatt tonight, must go on until we find out more and more about it. These specimens were nothing but granulation tissue covered by a single layer of flattened epithelium. In the tinted preparations it showed especially that it was made up mostly of granulation tissue with a lot of new blood vessels, and the supposed glands, to me, were nothing more than cystic cavities that looked something like pseudo-glands. They are lined with epithelium—but a very poor kind of epithelium. I would like to close by expressing my appreciation of Dr. Glatt's work in stimulating others to go on in this type of research.

Dr. Frank J. Novak: I wish to express my admiration for the type of work shown in this paper. Dr. Beck has covered a good many of the points I had hoped to discuss, so I shall omit much that I should like to talk about. The essayist presents a review of the management of chronic maxillary sinus disease which is at wide variance with the accepted views at the present time. We have long felt that the destruction either by disease or surgically of the lining of the maxillary sinus, finished it as far as that was concerned. There was no regeneration. Then observations were made which indicated that the mucous membrane did in some measure regenerate. Then with McGregor's study of twenty-two cases, I think, we all became convinced that the sinus mucosa may regenerate and in the regular way, after total and complete destruction. Now the study of these specimens points out that while the membrane may regenerate and usually does, it does not regenerate completely. Although the epithelial lining of the cavity may be of the normal stratified columnar type, the changes in the mucosa under the lining are not normal. We find in all these specimens an enormous increase in both cellular and fibrous elements of tissue. In the older specimens, in these individuals from which the specimen was taken after a considerable time, we find the granular tissue has changed in appearance until it has become a pure fibrotic mass with relatively few vessels, in other words, poorly supplied with blood. There are structures in the tunica propria which look like glands; they are not normal. They have a cystic quality. We must change our views on the basis of such study as this, that regeneration is complete and results in restoration to normal. Dr.

Glatt's view is to destroy the lining of the sinus and then forget about it—his plan is to prevent that regeneration instead of establishing drainage by a large opening to keep the ostium open. He is interested in blocking the ostium completely, in not providing accessory openings, and in filling the antrum with fibrous tissue or new bone, and that is what happens in cases in which he followed this method. Those in which an extensive fibrosis took place showed a clinically normal antrum. Perhaps it is purely theoretical consideration, but it is important here. When we open the sinus externally through the cheek we either remove the membrane entirely, meaning complete curettment down to the bone, or else a partial removal. If we remove it partially we will find such things as the formation of cysts. If we hope to replace the membrane with a tough fibrous skin we must depend on the formation of new bone. If we remove the entire lining and produce this complete denudation, we have then removed the only possibility by which new bone can be laid down. Bone has no capacity within itself to grow, except in the embryo, but in the adult bone if you remove the entire periosteum how can you possibly get generation of new bone? I think things must be modified to this extent. You must not remove the complete tunica propria. You must leave that adjacent layer of cells which has the capacity to change to osteoblasts and thus provide for the formation of new bone. I feel very kindly toward the whole proposition. It seems to me to be logical, and it seems to me that the effort to obliterate a sinus is something desirable, and while we can come to no final conclusion on the basis of a few slides and fifteen cases, I think it is a move in the right direction and should not only be carried on, but be fostered elsewhere so that we may have a comparison of results.

Dr. M. A. Glatt (closing): My statement about the healing of the middle ear cavity after a radical mastoid operation and the repair of skin after a total loss was not meant to be construed as an analogous process in the sinuses after a radical operation. My aim was to question the statements that are being made that the mucosa and the glands regenerate and are histologically normal, because one thereby implies that this reformed epithelium assumes an embryonic function.

In the sinuses one is dealing with rigid walls and since their collapse is disfiguring and dangerous, one must attempt to obliterate them with bone and scar tissue. I brought out the facts that the complete denudation of the mucosa without ventilation will help to bring this about. One should avoid curetting the bone, because one destroys with it the bone-forming cells that adhere to the bare walls. I also take exception to the curettage of the mucosa in the intranasal operations, because pieces of mucosa are left in the sinuses. In the

external approach, it is a faulty technique to leave pieces of mucosa because they do not appear to be diseased, or to desist from entering a recess.

I can see the value of ventilation in the acute, sub-acute and some chronic cases of sinusitis but not when the mucosa has been completely removed. The very large openings that are made in the form of "windows" or "doors," for drainage in such instances are of little value if not detrimental to the postoperative repairs.

Sewall on the other hand, deplores the obliterative process which takes place and advocates, following the regular Caldwell-Luc procedure on the maxillary sinus, the establishment of a permanent opening in the canine fossa. He admits that it is a nuisance to the patient, but the end results of a thin mucosa and very little obliteration of the cavity justify this as a routine procedure.

We, therefore, have now the two opposite points of view: Obliteration or nonobliteration. I believe that time will prove that complete obliteration is the most effective. As to the obliteration of the sinus ostium, I do not attempt it, but occasionally it will become obliterated. In the frontal sinus where the ostium is surrounded by bone, it is easily accomplished. In the maxillary sinus, where the ostium is in the membranous portion, it does not usually become obliterated. This was shown by the epithelialization which has occurred in those instances where the counter opening was not made into the nose.

Nasal Ionization: Biopsy Studies in Relation to Clinical Evaluation

A. R. HOLLANDER, M.D., AND NOAH FABRICANT, M.D.

(Authors' Abstract)

Since ionization was first introduced as a therapeutic procedure, it has passed through periods during which it has been enthusiastically recommended not only for nasal allergy but also for a large variety of other diseases. There has been manifested in some quarters a trend to reject ionization because histopathologic studies have allegedly demonstrated deleterious nasal tissue changes which were assumed to be permanent or requiring considerable time for resolution. Investigators generally have arrived at their results from single biopsies as controls, failing to take cognizance of the fact that the epithelium of the turbinates, for example, may vary in different sites. The present study has led to the belief that normal epithelium seldom remains normal in the classic sense, since epithelium, exposed to bombardment by multiple factors and conditions, is subject to change. There is an

urgent necessity for re-examining and re-evaluating the concept "normal" epithelium. In chronic infection superimposed upon an allergic process certain characteristic changes are produced which represent histopathologic pictures of both allergic and chronic inflammation of the infectious type. The two changes may vary in individual cases and even in parts of the same specimen. In view of the fact that the vast majority of specimens was obtained from either an allergic or a chronically diseased nose, it is quite obvious that the mucosa taken from such individuals has already undergone pathologic alterations. The problem presents itself: Does an allergic or a chronically infected nose afford a suitable "normal control" for the microscopic study of ionization? McMahon's and Smit's studies on animals are not applicable to human beings. Until biopsies made from the human nose are as complete in total surface, they do not adequately represent the complete pre- and post-ionization status of the human nose. The present method of histopathologic study of the nasal mucosa is open to question. All investigators have committed the same error. The biopsy method is too restricted to yield sufficient information about the entire nasal mucosa. Proper evaluation of the status of ionization with respect to vasomotor rhinitis and other nasal allergies must be obtained clinically rather than by microscopic investigation. The theoretic assumption that ionization produces harm in the nasal mucosa is not supported by histopathologic evidence. It can be asserted quite definitely that the electrocautery, drugs, chemicals, the x-ray, radium, surgical trauma and the like produce far greater insult to the nasal mucosa than does ionization. No histopathologic evidence has been produced to prove that nasal ionization is a harmful therapeutic procedure. Until such a time as more conclusive histopathologic evidence of the effects of nasal ionization is available, evaluation of the procedure should be based solely on clinical observation.

DISCUSSION

DR. FRANCIS L. LEDERER: Very frequently a therapeutic method which possesses some merit becomes popular because with it relief of symptoms can be afforded a certain group of patients who have gone through the gamut of specific and nonspecific remedies. This is precisely what has happened with ionization, for which, however, it must be said, the present authors have never claimed curative results for nasal allergy. Then there follows a piece of work by a certain group of laboratory workers and clinicians, and whatever enthusiasm has been gained or attracted is immediately lost, because harmful mucosal effects are shown to take place. The work, naturally, is accepted as conclusive, but must be reconsidered in the light

of a study such as has been presented by Hollender and Fabricant. If their contentions are correct, Dean's deductions are fallacious, necessitating a reconsideration of the ionization procedure and an evaluation of its merits purely along clinical lines as pointed out by the essayists. In my opinion this study is an important one, for even Dean is agreed that a certain percentage of nasal allergies not amenable to other measures can be relieved by ionization. We should not be too hasty in dispensing with a method until the evidence against it is decisively conclusive.

This work is referable to other procedures as well as to ionization and in this respect it emphasizes a phase of laboratory investigation which has not been properly considered in the past.

This paper then presents to me two valuable points:

1. That ionization must be judged solely on clinical results.

2. That biopsy studies of nasal mucosa with other procedures are open to question unless thoroughly supported by adequate histopathologic evidence. The single biopsy control is misleading.

Dr. L. B. Bernheimer: The essayist has spoken of evaluating ionization clinically. About a year ago, I published such a study, the results of which I would like to recall.

Twenty-five individuals suffering from hyperesthetic rhinitis and ten with seasonal hay fever were treated by zinc ionization and observed over a period of fifteen months. To compare the end results in this series with the results obtained with the use of such escharotics as phenol, trichloracetic acid, and zinc sulphate, twenty other individuals with hyperesthetic rhinitis and ten others with hay fever were treated with these chemicals.

Histologic observation of the affects of ionization on the nasal mucous membrane were found not to differ from those resulting from the application of escharotics. In neither instance was marked evidence of fibrosis found, the subepithelial stroma showing little tendency to thickening after resolution was complete. With both methods there were immediate changes in surface epithelium varying from complete destruction to simple inflammatory processes.

Clinically the course of hyperesthetic rhinitis was influenced in about 5 per cent of the patients treated with ionization, while slightly better results were obtained through the use of escharotics. The clinical course of hay fever was not altered by the use of either ionization or escharotics.

The most important conclusion drawn, however, was that ionization carries a definite risk of complications that do not present themselves through the use of escharotics. Two of the patients ionized developed anosmia, persisting for four months with one patient, and for seven months with the other. One patient developed an unilateral headache, typical of the syndrome discussed by Sluder, which persisted for three and one-half months. The majority of patients ionized complained of headache, sometimes associated with nausea, lasting for many hours after ionization. I have never encountered a complication following the use of escharotics.

My belief is that inasmuch as the relief of symptoms in allergic nasal disease is less striking through the use of ionization than with escharotics, and that since ionization carries risks of complications not encountered with escharotics, it is time the ionizing machine is dropped from the armamentarium of the rhinologist.

DR. FRANK J. NOVAK: There are several points that should be kept in mind. In the first place, what is normal epithelium or normal mucosa? We can see it only in the newborn, provided the newborn is not exposed to syphilis or other conditions in the mother. If he is normal the mucosa of the nose is normal. There is no adult in this climate who has not had changes in the nose. These changes are more or less constant and definite, and we know what they are. Therefore, it is fairly reasonable to accept the view that in an adult in this climate, exposed to the things we know he is exposed to, we can label the mucosa normal for that individual under these conditions, and we know what the changes have been. The changes in the epithelium will vary constantly. We know the stratified squamous epithelium is found behind the septum. We also know the posterior two-thirds of the nose, the lateral walls and the septum consist of normal pseudo-stratified epithelium. If we take a specimen from the anterior part in one case and the middle in another case we cannot compare the two. But in a series, if a specimen is always taken from the same place, we can, I think, say that that biopsy represents the normal. There are changes, as we know, which vary from normal. There is thickening in the membrana propria. We know it is composed, partly at least, of a certain type of fiber which has an affinity for silver. When they are separated by edema they have the appearance of a thickened membrana propria. We also know that there are changes in the submucosa, in the tunica propria. If these tissues were stained or impregnated with silver one could make a more or less quantitative estimation of changes in the tissue, both of the membrana propria and the tunica propria. Mention was made of the

various types of epithelium in the nose. There are only three types, pseudo-stratified, cuboid type and pavement type of epithelium. There is no transitional type here.

I am firmly convinced that while ionization may produce a deleterious effect on the nasal mucosa, I am also convinced that curettage, which we do so regularly, produces much more damage.

DR. SAMUEL SALINGER: I concede that Dr. Hollender is one of the outstanding proponents of ionization as a therapeutic agent and has had a vast experience which merits respect. However, it seems to me that far from strengthening his stand on this proposition his presentation this evening has greatly weakened his position and he has definitely taken a step backward when he states that the evaluation of the procedure will have to be made on clinical grounds. I had hoped and had even been led to believe that evidence would be forthcoming based on histopathologic studies which would prove that ionization was not only a beneficial agent but a safe one. This evening's presentation fails to offer any substantial refutation of Dean's and McMahon's work as to the effects of ionization on the mucous membrane. As a matter of fact Dr. Hollender admits that changes do take place but offers the alibi that similar changes are found in the tissues that have never been exposed to ionization. The important facts which we all want to know are, how much damage does ionization do, is the damage permanent and do the results justify the trauma inflicted? I hope that the authors will not be discouraged but will continue their studies in an effort to answer these highly important questions.

DR. J. A. WEISS: Has the essayist any information as to whether the ionized nasal mucous membrane ever becomes approximately normal in function by physiologic standards, such as the obsorption of active substances, for instance pituitrin, and, if so, how long after ionization may that be expected?

DR. NOAH FABRICANT (closing): I wish to thank the discussors for their comments on the paper. In mentioning the risks that follow ionization, anosmia, headache, nausea, and vomiting, which Dr. Bernheimer has had in his experience, I can say that in all our work at the University of Illinois we have not encountered such complications. Dr. Hollender says that he also has not encountered them. I myself have not seen any case of anosmia follow ionization at our clinic. We use the strip gauze method and we do not pack the nose too high. Possibly such cases may occur, and perhaps it has been only our good luck that we have not had them. I can imagine that

through faulty technique such things might happen. Whether escharotics are best or ionization is best, is not a problem that comes under out title. I know escharotics have proved of value, but I feel that ionization has a place in our therapeutic armament. We ask the same trial for ionization that is given patients following an allergic survey. It has been mentioned also that escharotics should be prescribed and ionization dropped. But I think it can be admitted that escharotics also have their failures. Ionization has had a degree of success in various hands, as well as failures.

Dr. Novak suggested that in studying biopsy specimens, the method should be to take the specimen from the same point each time. I feel we covered that point and illustrated that weakness, for the reason that if you take a specimen from a certain area before ionization and then take another afterward, you have trauma before ionization and trauma after ionization. There is resultant scar tissue. Then if you take a specimen from the same site you have trauma piled on trauma, and I am afraid you would be misled.

The question was asked whether studies in physiologic stains have been made. They have not been, and I think it is a problem for future work that merits consideration. Dean's "The Fundamentals of Allergic Rhinitis with Particular Reference to Ionization" is more optimistic than I am. He makes the statement that ionization does improve most cases of allergic rhinitis. I feel it may improve many, but not most. Then McMahon makes this statement in the same symposium upon allergic rhinitis: "There should be some place in our therapeutic armamentarium for a method for which such beneficial clinical results are claimed." If the tissue changes are so deleterious, let us not employ ionization; if they are not, let us give it a trial. I do not feel one should say they are dangerous, and then use the method. If the tissue changes are only partially damaging, I would say such a position is feasible.

Books Received

Surgical Anatomy of the Head and Neck.

By John Finch Barnhill, M.D., F.A.C.S., LL.D. Formerly Professor of Otolaryngology in the Indiana University School of Medicine. Emeritus Professor of Surgery of the Head and Neck. Formerly Chief of the Department of Head and Neck Surgery in the Indiana University Hospitals, Honorary Professor of Anatomy, University of Southern California School of Medicine. Introduction by Paul S. McKibben, Professor of Anatomy in the School of Medicine, University of Southern California. Cloth. Royal 8vo. of 921 pages, with 431 illustrations. Baltimore: William Wood & Company, 1937. Price $20.00.

This book appears to be a collection of notes, probably taken stenographically from Doctor Barnhill's lectures. It is to be regretted that the work did not have more extensive editing before being put into book form. There are many typographical errors, misspellings and omissions. Many of the illustrations also are so crudely drawn as to be of little informative value.

Most of the material is presented in the first person, which makes it too redundant for consecutive reading and at the same time diminishes its value as a reference work.

The illustrations are all at the back of the book, making it necessary to keep the book open in two places, a patent disadvantage if it is to be used as a dissector's manual.

Doctor Barnhill's lectures have long been popular for their forceful presentation and practicality and merit a more careful publication than they have received in this work.

A Textbook of Ophthalmology.

By Sanford R. Gifford, M.A., M.D., F.A.C.S., Professor of Ophthalmology, Northwestern University Medical School; Attending Ophthalmologist, Passavant Memorial, Cook County, Wesley Memorial and Evanston Hospitals. Cloth. 492 pages with 249 illustrations. Philadelphia and London: W. B. Saunders Company, 1938. Price $4.00.

A small compact handbook which should prove useful to the laryngologist as a ready reference work. Capably presented and profusely illustrated.

Abstracts of Current Articles

NOSE.

Voluminous Left Ethmoidal Chondroma With Pan-Sinus Mucocele and Severe Ocular Symptoms (Voluminoso condroma etmoidale sinistro con mucocele pan-sinusale e grave sindrome oculare).

Rubaltelli, E. (Univ. of Padova), Riv. Oto-Neuro-Oftal., 14:48 (Jan.-Feb.), 1937.

A healthy 16-year-old carpenter, for about five months previous to his admission to the clinic, had noticed a progressive external deviation, a gradual exophthalmus and marked loss of vision of his left eye. On palpation, a hard, fixed and painless mass was felt at the superomedial portion of the left orbit. Anterior rhinoscopy disclosed a profuse mucocatarrhal discharge, slight enlargement of the inferior turbinate, mucosa over the anterior margin of the middle turbinate appeared thinner and more gray than normal. The superior part of the middle meatus was examined with a probe and a hard, painless, immovable mass was felt. On transillumination a marked opacity of the ethmo-orbital region of the left side was visible. Roentgen plates revealed an opaque mass of the size and shape of a walnut, smooth inferiorly and irregular superiorly, occupying the region of the left ethmoid cells.

Due to the extremely high position of the tumefaction, the author decided in favor of the transmaxillary or centrofacial approach of Citelli instead of the transmaxillonasal approach of Moure.

Local anesthesia was employed. This large eburnated mass was found to be free at the inferior margin and tightly adherent but not continuous with the orbital and maxillary sinus walls, but superiorly it was fused to the bony tissue of the frontal vestibule and naso-frontal duct. At this latter location a chisel had to be used with extreme care to remove the growth in toto and avoid entering the cranial or orbital cavities.

The frontal and maxillary sinuses had to be opened to remove aseptic mucoceles which came into view while the tumor was displaced.

The patient recovered completely and the cosmetic result was good when seen after 17 months.

Rubaltelli discusses the etiology, pathology, symptomology, diagnosis and frequency of recurrence of nasal chondroma. He further

reviews the indications and advantage for employing the various techniques for removal of nasal growth and emphatically states that Citelli's is the best suited and less mutilating when operations are to be performed in the uppermost region of the nasal cavity and upper nasal sinuses.

SCIARRETTA.

Tumors of the Orbit: Cylindroma of the Lacrimal Gland (Tumori dell' orbita: Cilindroma della ghiandola lacrimale).

DiMarzio, Q. (Bologna), Riv. Oto-Neuro-Oftal., 14:65-108 (Jan.-Feb.), 1937.

Protocols, numerous photographs and photomicrographs of seven cases of cylindroma of the lacrimal gland are presented. Two of them had recurrence. Case No. 4 was reoperated after 20 years and three years later died from endocranial metastases. The other, case No. 5, after the third recurrence, was treated by surgery and röentgentherapy with no reappearance of the tumor after eight years.

The author discusses, at length, the opinions of many American and foreign writers as to the etiology, pathology and terminology of this pathological entity. He, personally, believes that these tumors are of mixed tissue type and originate from various embryonic layers because some have endothelial and other epithelial characteristics. Such findings, he thinks, may be explained if one considers the manner in which the lacrimal gland and its capsule are formed during the embryonic period. That is, the epithelial elements, which are to form the tubules of the glands, leave the conjunctiva and, before reaching their destination, pass through the mesenchyma which is destined for the various types of connective tissue. It is probable, therefore, that these epithelial masses may drag with them some mesenchymal tissue. These mesenchymal germs, maturing later on, give origin to a variety of connective tissue, within which masses of endothelial tissue or glandular tissue of embryonal type become incarcerated.

To explain the presence of various types of connective tissue it is also necessary to note that they develop in close contact with the periosteum of the orbit; that is, with a tissue possessing great proliferative activity and metaplastic ability. Therefore, it is not surprising to find, in cylindroma of the lacrimal gland, metaplastic tissues of cartilagenous or mixomatous type.

The author advises early and total removal of the tumor and the lacrimal gland followed by appropriate röentgentherapy.

SCIARRETTA.

Contribution on Mucous Cyst of the Nasal Vestibule (Contributo allo studio delle cisti mucoidi del vestibolo nasale).

Giuffrida, E. (Univ. of Catania), Arch. Ital. di Otol., Rino and Laring., 49:71 (Feb), 1937.

A case report of mucous cyst in the right nasal vestibule is presented and the histogenesis of this pathological entity is discussed by the author.

He states that while most investigators agree that these cysts are the result of an embryonic developmental disturbance; yet a close analysis of each case report brings out a great variety of histogenetic hypotheses, and these he groups as follows:

The first theory suggests that mucous cysts originate from aberrations of cellular masses during union of the facial fissures. This is supported by Klestadt, Kelly, Beck, Aubriot and Citelli.

The second claims origin from the nasopalatine duct from rests of Stenson's canal, presented by Grünwald, Malan and Liveriero.

The third, suggested by Brüggemann and Carcò, claims that the cyst originates from the nasolacrimal duct.

Giuffrida discusses the above and many other hypotheses and believes that the histogenesis of these malformations is the result of aberrations of entodermal masses developing when fusion of the facial clefts takes place. The constant location of this cyst (the external portion in the floor of the nasal vestibule) induces him to believe that this inclusion takes place when the superior maxillary process of the first branchial arch fuses with the nasal process.

Mucous cysts of the nasal vestibule may be, genetically, considered similar to branchial cysts of entodermal inclusions, admitting that in this location entodermal inclusions may be present and develop into mucous cysts; same as ectodermal inclusions give origin to dermoid cysts.

SCIARRETTA.

A New Principle for the Treatment of Ozena (Un nouveau principe de la thérapeutique de l'ozène).

Dorochenko, I. T., Acta Otolar., 25:542 (Nov.-Dec.), 1937.

Intramuscular injection of 1 cc. of "gravidan," a product secured from the urine of pregnant women, containing prolan, folliculin and other hormones, produced marked improvement in 28 cases, apparently through sympathetic stimulation.

FENTON.

PHARYNX

Tuberculous Ulcerations of Mouth and Pharynx.

Ormerod, F. C. (London), J. Laryng. and Otol., 52:675 (Oct.), 1937.

This is an account of ten cases of such ulcers treated by the author. He has tried lactic acid, guaiacol and trichloracetic acid but regards high frequency cauterization as the best treatment in the mouth and pharynx. He prefers the actual cautery for the larynx.

The high frequency current is applied under cocain anesthesia, using a needle electrode for the edge of the ulcer and a ball electrode for the base.

Pharyngeal Paresthesis and Their Treatment at Thermal Resorts (Les Syndromes paresthesiques pharyngés et leur traitement thermal).

Courbin, J. (Bagnères), Rev. de L. O. R., 58:866 (Sept.-Oct.), 1937.

This careful analysis of painful pharyngitis and its therapy calls attention to the necessity of accurate metabolic and systemic study, even occasional neuropsychiatric investigation. In view of the pending study of American thermal establishments by the American Medical Association, the adjuvant effects stated for various French mineral springs are of interest, particularly sulphur, for chronic inflammatory conditions and calcium and sulphur for rheumatoids. The effects of climate, change of environment, and of various baths and therapeutic applications at these resorts are thought to do much for the neurotic types of paresthesias. If excessive claims and undue expense can be obviated, American resorts may regain some of the prestige which now accrues to European spas.

FENTON.

ESOPHAGUS, TRACHEA AND BRONCHI

Alcohol and Cancer of the Esophagus (Alcool et cancer de l'oesophage).

Piquet, J. and Tison. Bronchoscopie, Oesophagoscopie, et Gastroscopie, April, 1937, p. 137.

The authors reviewed the statistics in 110 cases of esophageal cancer and found a history of habitual use of alcohol in 93 to 95 per cent. Nine of their cases were in women and 101 in men, an overwhelming proportion of men which is not repeated in statistics from other countries, and which they believe is due to the fact that the regular use of alcohol by women is exceptional in France.

Furthermore, 82 of the cancers in alcohol users were located above the bronchial bifurcation, while only 20 were located below this level. This proportion differs from the usual statistics and is due,

the authors feel, to the relatively more intense exposure to alcoholic irritation of the upper end of the esophagus, with a resultant tissue metaplasia in this region. They think that alcohol must be classed as an important cancerigenic agent in susceptible individuals.

KLAWANS.

Endoscopic Operation on a Large Pulsion Diverticulum (Opération endoscopique d'un gros diverticule de pulsion).

Seiffert, A. Bronchoscopie, Oesophagoscopie, et Gastroscopie, July, 1937, p. 232.

In a markedly emaciated individual with a very large esophageal pulsion diverticulum of 30 years standing, the author operated through an esophagoscope by dividing the wall between the sac and the esophagus over a distance of 5 cm., establishing a wide communication.

There were no complications, and within a few weeks the patient gained twenty pounds in weight and was relieved of all symptoms. Seiffert recommends the procedure as simple and safe in similar cases with enormous diverticula and thin party walls, and feels that it might even be applied in smaller diverticula.

KLAWANS.

Cardio-phrenic Esophageal Stenosis (Le rétrécissement cardio-phrénique de l'oesophage).

Soulas, A. Bronchoscopie, Oesophagoscopie, et Gastroscopie, April 1937, p. 141.

In discussing in great detail so-called cardiospasm, with seven case reports, the author denies the role of spasm either of the diaphragmatic crurae, or of the cardia as an etiological factor. He points out that the obstruction is not at a localized point, but extends over a considerable area, so he prefers the term "cardiophrenic stenosis."

His studies lead him to conclude that practically every such case is the result of inflammation, an esophagitis or peri-esophagitis, with secondary scarring of the muscularis and muscularis mucosae layers and consequent narrowing with subsequent dilatation above the obstruction. This dilatation differs from true mega-esophagus, which is a different condition.

Treatment, if the patient is seen early, consists of medical measures and careful dilatation under direct esophagoscopic guidance. Blind dilatation is condemned. In old or resistant cases, he advises operation by extramucosal cardiotomy or esophagotomy which he considers a simple and safe procedure which produces excellent results.

KLAWANS.

Vascular Adenoma of the Bronchus.

Zamora, A. M., and Schuster, N. (London), J. Laryng. and Otol., 52:337 (May), 1937.

In this article the authors review forty-two cases of benign tumors of the bronchus which may be grouped under the classification "vascular adenoma." The paper emphasized the microscopic characteristics of the tissue in these cases, and points out that benign tumors of the bronchi are not so rare as they were once thought to be.

Two cases are reported in detail.

Technique of Obtaining Separate Direct Intrabronchial Smears for Tubercle Bacilli (Technique du prélèvement direct intrabronchique du bacille de Koch et division des crachats).

Lapine, G. Bronchoscopie, Oesophagoscopie, et Gastroscopie, July, 1937, p. 181.

The author uses the flexible, radio-opaque, two-way flow catheter of Hicquet, about 45 cm. long, 6 mm. caliber, and with pointed ends. Anesthesia is obtained by using 2 to 4 cc. of 2 per cent cocain in cherry laurel water, with adrenalin and phenol added, instilled through the nose with the head bent backwards. The same solution is also instilled into the trachea. The catheter is passed through the nose and introduced into each main bronchus in turn under fluoroscopic guidance. No lubricating oil is used, as this may interfere with the staining of the smears. Ten cc. of normal saline or of distilled water is now injected through the catheter, and after several minutes, 2 to 5 cc. is aspirated and collected in a sterile tube. This washing may be repeated. The opposite side is then treated in the same way and separate specimens obtained.

By placing the patient in a lateral decubitus position the upper lobe bronchi may be entered and separate specimens obtained. The method is simple, well tolerated, requires minimum anesthesia, and has given good results in the more than one hundred cases in which it was used.

KLAWANS.

Röle of Helium in Cases of Obstructive Lesions in the Trachea and Larynx.

Kernan, John D. and Barach, Alvan L. (New York). Arch. Otolaryng., 26:419 (Oct.), 1937.

The physiologic basis for the use of helium in cases of obstructive lesions in the trachea and larynx is presented. The clinical results of inhalation therapy with the use of helium and oxygen in twenty-one cases are described. In some patients tracheotomy has

been averted by this procedure; in others the condition of the patient has been improved, tracheotomy having been required later. In still other cases, in which the obstruction was very marked, helium and oxygen therapy was of no avail. The variable factors which determine the clinical application of this method of treatment are discussed.

TOBEY.

EAR.

Use of Urea in Treatment of Chronic Otitis Media (Preliminary Report).

Mertins, Paul S., Jr. (Montgomery, Ala.). Arch. Otolaryng., 26:509 (Nov.), 1937.

The author quotes in part much of what Holder and MacKay said: That urea in aqueous saturated solutions or in crystalline form by its power of dissolving necrotic debris removes the chief deterrent to healing in many types of chronic aural infection.

It is almost totally nonirritating to living tissue.

It is mildly bactericidal to many forms of organisms, especially those producing putrefaction.

It almost immediately eradicates all odor without producing another odor.

It is inexpensive.

TOBEY.

On a Case of Deaf-Mutism of Traumatic Origin.

Hallpike, C. S. (London), J. Laryng. and Otol., 52:661 (Oct.), 1937.

This is an account of the changes revealed by histopathological examination of the temporal bones of a patient who, being described as deaf-mute from early childhood, came to postmortem examination at the age of 59 years by reason of an acute urinary infection. The patient was described by relatives and friends as having always been deaf, although this had been definitely established at three years of age. The patient was not known to have suffered any head injury at any time.

The right temporal bone, however, was found to have been fractured transversely through and involving the greater part of the petrous pyramid. On the left side, no fractures or other abnormalities of the labyrinth capsule were found. Extensive degeneration of the cochlear and vestibular end-organ was, however, present.

In a series of 36 photomicrographs, the exact pathological changes in both temporal bones are presented.

Syphilitic Mastoiditis—Operative Indications (Indications operatoires des mastodites syphilitiques).

Leroux, L. (Paris), O. R. L. Internat., 21:407 (July), 1937.

Pointing out that painful mastoid swelling due to syphilitic periostitis rarely shows any tympanic involvement, Leroux rejects the idea that surgery should never be done in the presence of deep syphilitic osteitis. Secondary purulent involvement of the luetic lesion will occasionally necessitate extensive operation, but must obviously be accompanied by intensive antisyphilitic treatment. Any painful mastoid swelling without loss of hearing should suggest serological tests.

FENTON.

Observations on the Semicircular Canals.

Peet, E. W. (London), J. Laryng. and Otol., 52:431 (June), 1937.

The purpose of this paper is to show that the anatomical relationship of the semicircular canals of one ear to those of the other is inconstant. In the skulls of two children, the angle between the axis of the superior canal on one side and the posterior canal on the other side is 41 degrees. In four adult skulls this angle varied from 14 to 20 degrees.

Hitherto Unrecognized Danger in Ramadier's Operation for Petrous Suppuration.

Guild, S. R. (Baltimore), Acta Otolar., 25:561 (Nov.-Dec.), 1937.

Sections of the temporal bone in five individuals, one bilateral, from 26 to 65 years, exhibit patches of definite adhesion between the thinned carotid wall and its bony canal. These would inevitably result in tearing the artery in any paracarotid approach to the petrous apex, such as those of Lempert or Ramadier.

FENTON.

Clinical Observations on Bone Conduction.

McNally, W. J., Erickson, T. C., Scott-Moncrieff, R. and Reeves, D. L. (Montreal), J. Laryng. and Otol., 52:295 (May), 1937, and 52:375 (June), 1937.

This is a report based upon the bone conduction tests made in the course of the examination of fifty patients in whom an intracranial lesion had been proven at operation or at autopsy.

The instruments and tests are described in detail. Owing to the difficulty in standardizing any one of these tests, the summary is based upon the conclusions reached through a number of them. These are too diverse to lend themselves to concise abstraction.

Otitis Media in Early Childhood (Under Five Years).

Discussion before the Royal Society of Medicine—Section of Otology, May 7, 1937. J. Laryng. and Otol., 52:685 (Oct.), 1937.

A detailed discussion of the subject by five members of the Society with case reports, tables of incidence both of the disease and of the organism involved. The occurrence of diarrhea and vomiting in association with otitis in infants was stressed.

Notices

An intensive refresher course in operative surgery on the cadaver will be given May 16 to 26, inclusive, by the Departments of Otolaryngology and Anatomy in the Medical College of the University of Cincinnati. Applicants will have abundant material on which to perform various standard operations on the middle ear, mastoid, accessory sinuses, as well as some neck and laryngeal surgery. The instruction is given chiefly by the Staff of the Medical College and the Cincinnati General Hospital.

The Eighth International Congress for the study of defects of speech and voice will be held at Salzburg July 28 to 30, 1938. The official languages of the Congress will be German, English, French and Italian. Currently there will be held an exhibition of phonetics. The Secretary is Dr. Desider Weiss, Wien, IX, Ferstelgasse 6.